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# Archives of Neurology and Psychiatry

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## NEUROLOGICAL SURGEONS: WITH THE REPORT OF ONE CASE \*

HARVEY CUSHING, M.D.

BOSTON

The introduction of india-ink into the fluid spaces surrounding the central nervous system to see where the granules lodge has been an experiment repeated by many of us. We know that it blocks the normal circulation as effectually as does a granular fluid introduced into the spaces of a fountain-pen. In like fashion not a few of us, with little originality, periodically repeat the experiment of putting our calamus scriptorius to paper, despite the fact that the written word may sometimes choke rather than facilitate the circulation of ideas. Some of us, indeed, on scanning our former compositions are conscious that much of them would better have been writ in something less indelible than ink; and so, as we grow older this business of writing, particularly under compulsion, comes to be looked on with no little misgiving.

Nevertheless, one cannot so far depart from tradition as to ignore a time-honored obligation, and custom has it that your presiding officer should justify himself so far as he may be able by the discussion of some subject on which his attention at the time may happen to be focussed. Since I seem to have been the first surgeon admitted to membership in this Association and certainly the first to occupy the chair, my theme should properly be a neurosurgical one and it seems inevitable that it should concern brain tumors, for there is no other subject on which our varied interests are more likely to meet as on common ground.

Instead of inflicting you, however, with a formal discourse, a more objective method of bringing to your attention some aspects of this consuming topic will be employed. One cannot philosophize from an armchair in regard to surgical matters as he can about the diagnosis, classification and treatment of mental disorders; and consequently some of the patients with proved or suspected tumor who happen to be in the hospital wards at this time will be presented for discussion in another session.<sup>1</sup>

\* Presidential address at the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, May 31, 1923.

1. A report of this clinic will appear in a subsequent issue.—Ed.

I am aware that this is not a clinical society, and that I am proposing an innovation, but it is highly desirable that the Association as a whole, so long as it has admitted neurosurgeons into its membership, should have some idea of the varied character of the neurologic material which tends at the present day to gravitate into a surgical clinic, and of at least one of the major problems there being faced. For though the technical details of intracranial operations for tumor are of interest to only the few, the preliminary diagnoses which have led up to these measures, and their after-results, cannot fail to be of concern to every one of us.

It may be assumed that we are all engaged in problems of one sort or another relating to disorders of the nervous system because we have come to feel the special lure of this department of medicine. It makes little difference whether we have become sensitized to the subject in its sociological, its experimental or its clinical aspects; once successfully inoculated we are lost. For most of us, be we called neurologists, psychiatrists, or neuropathologists, this observation is a commonplace; but for those who have newly emerged from general surgery and have taken up neurosurgery as a specialty it is a novel sensation to find themselves in the grip of a subject which makes the more time worn forms of surgery appear to be somewhat tame and unintellectual affairs—ones which present no comparable attractions and offer no comparable difficulties.

That surgeons should have been admitted into this intimate guild now nearing its half-century of existence speaks well for the open-mindedness of its members. And if any of you had misgivings as to our acceptability here, on the basis that metaphysicians and artisans make an impossible social mixture, we can only hope that time will justify your action by our becoming through this contact better neurologists and your becoming more familiar than before with the possibility of handicraft as an increasingly dependable therapeutic measure for certain otherwise hopeless maladies. The fusion will be of benefit if for no other reason than that the balance sheets of surgery should periodically be audited by those not actually engaged in its practice.

It would be interesting to know what influences have severally led each of us into this general pasture of neurology and through it into our own special fence-corner. One case at least I can report on. It shows that chance and opportunity often counteract inclination. The story can best be given in the words of the patient himself.

The medical traditions of my forebears inclined me to the abnegating life of a family practitioner. To this I looked forward during my medical course in the early nineties; and I still think there is no more satisfactory or higher calling in medicine. But providence willed otherwise.

As an undergraduate I had attended with no especial thrill what for the time must have been an excellent series of clinical exercises in neurology. I can recall, strangely enough, that in this course I saw for the first time a case of exophthalmic goiter, and another of acromegaly, and heard leontiasis ossea and sporadic cretinism discussed.<sup>2</sup> But on the whole, the impression was gained that the diseases of the nervous system were obscure and included chiefly those maladies for which little could be done—maladies, in short, in which the profession as a whole showed little interest.

I had, to be sure, used as a text-book that admirable small treatise on nervous diseases written by Christian A. Herter, himself a victim of one of them; but this book was devoted purely to diagnosis and we students were given no opportunity to thoroughly study for ourselves and to examine from day to day an individual patient with a nervous disorder. Furthermore, we gathered from our remote position on the benches that treatment could be summed up under bromids, iodids, electricity, and the asylum.

Such minor operations as were recommended were largely orthopedic in nature, and though the skull might often enough be trephined for fractures, I saw as a hospital intern in surgery only one operation that I remember for an organic lesion of the brain. A patient with jacksonian epilepsy having a "march" involving the upper extremity was sent into the hospital for operation, by the professor of neurology. After repeated craniocerebral measurements the situation of the chief fissures was marked with an indelible pencil on the shaven scalp, the precise spot overlying the center whence movements of the thumb were presumed to originate being indicated by a cross. At this spot the surgeon, into whose service the case had come, was expected to trephine, and on the eventful day, through a stellate incision he removed a button of bone about an inch in diameter. A discussion ensued as to whether the tense dura which had been exposed should be incised, a step which was finally taken without disclosing a recognizable lesion. This negative finding was looked on as rather a joke on the neurologist, who, poor man, had assumed the entire responsibility, and there seemed no reason for any further surgical interest in the matter.

This was a typical example of surgery made to order, a relic of medievalism in medicine brought down to the end of the nineteenth century. To every onlooker the only way to avoid such an impasse must have been obvious. Either the surgeon would have to take greater interest in the problem through familiarizing himself with the brain and its diseases, or the neurologist would have to learn enough surgery to do the operation himself. It is not entirely clear which of these eventualities has been responsible for the changes time has brought about.

This story, which could probably be reduplicated by all of you who remember the neurological surgery of the nineties, is told because it illustrates a relationship between neurologist and operator as ineffective as it was intolerable. The surgeon concerned was one of the most brilliant, courageous and successful

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2. The neurologic aspects of the endocrine disorders in those days so far outweighed their metabolic and chemical aspects that they were included for the most part in the category of nervous disorders. And though one by one, with the exception of the hypophysis, the ductless glands have since been lost to us we may take pride in the fact that meanwhile some of the fundamental discoveries relating to their morbid states were made by neurologists.

operators of his day—a man who contributed greatly to abdominal surgery for the sole reason that in the diagnosis of lesions that lie between wind and water he was able to arrive at and to act on his own independent opinion. In the operation described he took no responsibility but acted merely as an unwieldy tool in the hands of another person.

Soon after I went to Baltimore in 1896, a patient with paraplegia due to a recent gunshot wound of the neck was admitted to Dr. Halsted's service and, as chance would have it, to the ward under my care as a house officer. At the time, the hospital had no roentgen-ray department, but with the aid of a huge static machine (relic of the neurotherapeutics of the day) and a small Röntgen tube no bigger than a baseball, I ground out, after repeated trials, enough roentgen rays in the course of prolonged twenty-minute exposures to cast on a photographic plate the shadow of a bullet lodged in the body of the sixth cervical vertebra. You may judge from this that she was a cooperative patient, and though at the time I was much more absorbed in Röntgen's recent discovery than in neurology, and had never before heard of hematomyelia, for the next six months with such precision as I could master I plotted out her anesthetic skin fields and followed daily with accumulating interest her subsiding paralyses.

This opportunity personally to study for the first time a neurologic case, which though a nonoperative one had drifted into and was permitted to remain in a surgical ward, made a great impression on me, and, spurred on by H. M. Thomas and L. F. Barker, with the report of this case I started on my inky way.

A surgeon, however, need not even then have apologized for writing on a purely neurologic subject. There was ample precedent. It was the accompanying paralysis that interested Percival Pott, not the spinal caries and resultant kyphosis. Moreover, in reading round the subject of hematomyelia in the nineties it was evident that the two most important monographs dealing with compression of the spinal cord had been written by surgeons—by William Thorburn and by Theodore Kocher. Here, unquestionably, were men who, making no pretense to be anything other than general surgeons, yet were interested no less in the neurologic than in the surgical aspects of spinal disorders.

But cerebral surgery at the time was in the doldrums. Many surgeons, to be sure, during the preceding decade which had practically seen the end of serious postoperative sepsis, had been encouraged to undertake a variety of operations for a number of cerebral disorders. It proved, on the whole, a disheartening business and for the most part these were very sporadic efforts made at the solicitation of neurologists,<sup>3</sup> neither party at the time fully appreciating the technical difficulties the procedures involved. It was still the day of the mallet and chisel for entrance; of wound drainage for closure; and a fungus cerebri with ultimate infection was the almost inevitable and horrifying consequence of most interventions for tumor.

Twelve years before, in the early eighties, following the publication of Ferrier's treatise on the localization of cerebral function, what appears to have

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3. For example, on October 25, 1895, a meeting was held in Boston to discuss the general topic of "The Present Status of Brain Surgery." Four neurologists participated under the following heads: Trephining for Insanity (Walter Channing); Trephining for Epilepsy and Infantile Cerebral Palsy (Morton Prince); Trephining for Cerebral Haemorrhage (George A. Walton); Trephining for Tumor and Abscess of the Brain (Philip Coombs Knapp).

been the first successful attempt to put the results of his observations actually into play was when on Nov. 25, 1884, under the direction of Hughes Bennett, Godlee exposed and partially removed a subcortical glioma from the arm center of the motor cortex. Anyone who wishes to gain some conception of the advances which have since been made in the surgery of brain tumors will do well to read the detailed account of this early venture and the discussion which it aroused—a discussion, be it said, which was participated in not only by Hughlings Jackson and Ferrier who were rather pessimistic, but by a young man of twenty-seven years named Victor Horsley, then engaged in some physiologic researches with E. A. Schäfer, and who warmly recommended morphin as a means of controlling hemorrhage. There was also present from Glasgow the towering figure of Macewen whose masterpiece, however, on the Pyogenic Infective Diseases of the Brain did not appear till eight years later.

During the intervening decade, though "trephining" for one object or another had been enthusiastically broached, the results of these procedures, when compared with those in other fields of surgery rapidly being opened up, were too discouraging to prove more than a temporary attraction for the surgeon. Evidently an entirely new operative technic would have to be developed before any distinct progress could be made—a technic which involved wider exposures of the brain, the control of hemorrhage as well as of tension and edema during the performance, and an absolutely secure wound closure at the end.

In 1893, the same year which saw the appearance of Macewen's monograph, there was published in this country a notable volume on "Brain Surgery," which begins with this paragraph:

"There are two essential preliminaries to any operation upon the brain. The first is the diagnosis of the nature of the disease which is present, and the second is the diagnosis of its situation. Both are purely medical questions, and until they are decided the surgeon cannot be asked to operate."

I am confident that if Allen Starr, in view of his position in neurology and his interest in surgical matters, had taken to the scalpel rather than the pen we would now be thirty years ahead in these matters, and I am sure his fingers must many times have itched when he stood alongside an operating table and saw the operator he was coaching hopelessly fumble with the brain. Indeed it was not until fifteen years after the publication of this book that he wrote in regard to an operation for an acoustic neurinoma on one of his patients that it was the first really successful tumor extirpation in his records. It is evident from all this that the surgeon long played a very secondary rôle and acted only under the instructions of the neurologist. There is no wonder, therefore, that the period was one of utter discouragement during which, except by one or two enthusiasts, the comparative futility of most of these operations came to be emphasized.

But in the foreground of this gloomy picture there stands out a gladiatorial figure—that of Victor Horsley. He alone of all these surgeons had been sufficiently fired by the spirit of neurologic research to give time to the experimental laboratory. In January of 1886 the Neurological Society of London had been founded with Hughlings Jackson as its first President, and with Horsley, though primarily a surgeon, as one of the original members. So from the first he sat with his peers, and when a month later (Feb. 9, 1886) he was

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4. Bennett, A. H., and Godlee, R. J.: A Case of Cerebral Tumor, *Med. Chir. Trans.* 68:243, 1885; also *Proc. Roy. Med. and Chir. Soc. of London*. N. S. 1:438, 1885.

appointed surgeon to the National Hospital for the Paralyzed and Epileptic, Queen Square, the birth of modern neurologic surgery may properly be assumed to have taken place.<sup>5</sup> Though work came slowly, it was in that hospital only a year later (in June, 1887), when he performed for Gowers an epoch-making operation—the first extirpation of a tumor from within the spinal canal—an operation which he alone possibly of all living surgeons was capable of seeing to a successful issue because his surgical training had been supplemented by laboratory experiences which had familiarized him with comparable operations on the nervous systems of animals. It was a fortunate beginning, one which helped greatly to offset the conservative attitude widely held in regard to interventions of this sort. Neurosurgery at this time needed a fighting champion, and such an one he was—in this as in other causes.

When, in 1900, at the termination of a long surgical residency at the Johns Hopkins I went abroad for a year, it was with the purpose of working on some neurologic problem; and having become somewhat surgicalized in the course of the preceding five years I knew no better than to seek someone of my own craft as the source of inspiration. Naturally my steps turned to London, where I found Horsley kindness itself, but despite an insatiable craving for research his time had become so taken up with a large consulting practice, with the development of his general surgical service at the University College Hospital, as well as with matters relating to professional politics, that most of his investigative work was done at odd hours at his home. As Stephen Paget, his biographer, says, his period of incessant laboratory activity which had begun in 1884, was then about at its close.

So with vague talk of returning for some work later on, I drifted to France and finally to Switzerland where in Berne I passed the most engrossing year of my medical life in Kronecker's laboratory on a problem connected with intracranial tension—an *Arbeit* which Kocher had suggested. This was followed by an all too brief period with Sherrington who was then in Liverpool, where I was permitted to participate in some experiments on the delimitation of the motor cortex of anthropoids.

Thus equipped, with my sole neurologic qualifications represented by these two experiences, I returned to Baltimore and petitioned for a post as neurosurgeon in the clinic.

My former chief was evidently staggered at the proposal. He suggested as an alternative that I take a position in orthopedics, a subject which in his opinion covered practically all neurologic maladies having surgical bearings. To be sure, there were the neuralgias, but brain tumors of which there had been only two or three examples in the decade since the hospital opened were too uncommon to be considered.

In short, there was no possible source of livelihood in neurologic surgery and did I know of anyone, even Horsley, who had actually limited himself to

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5. Just twenty years later (July, 1906) at the Toronto meeting of the British Medical Association, Horsley gave in detail his views regarding the technic of operations on the central nervous system—a subject in which great advances have since been made. His temperament was incompatible with the time-consuming and fastidious details of technic which most of the neurosurgeons of the succeeding generation regard as essential to the success of operations for tumor. In this address he dwelt largely on chloroform as an anesthetic and on the treatment of shock. General anesthesia is now giving way to local, and operative shock is the least of our present troubles.

any such specialty? I did not. And I recall with embarrassment making the suggestion that exophthalmic goiter might be regarded as a primary disorder of the nervous system: operations, indeed, on the cervical sympathetics had been undertaken, and in Berne they were beginning to operate on the gland itself. But this met with no favor and my neurologic prospects seemed far from encouraging. Consequently, for the next few years I continued to do general surgery in an adjacent hospital, and contrary to all expectations, neurologic cases began to accumulate in such numbers as to guarantee a living. Meanwhile unlooked for laboratory facilities were secured and I was soon able to devote myself exclusively to operative neurology and the laboratory.

We are all familiar with patients who are led into numerous and somewhat lengthy digressions in the recital of their past history, and the story just given proves no exception. The record, moreover, is fragmentary. It would have been more in keeping with custom had mention been made at the outset of the patient's chief complaint; moreover, his present status should be thoroughly gone into and at the end a therapeutic program should be outlined. The *complaint* is an imperfect elementary training in neurology, and though it is too late for this to be remedied in the particular instance cited, a proper treatment which may prevent similar complaints in the future on the part of others, can at least be outlined.

Unquestionably, if the future neurologic surgeon is to do the thing properly, his training will demand a longer preparation than that needed for any medical specialty—and not many will have the industry, the patience, or the intellectual gifts combined with the manual dexterity, necessary to see it through.

The essentials are a far more thorough grounding in the structure, function, and morbid anatomy of the nervous system than is provided in the preclinical years of any of our schools, and with far more attention paid to the diseases of the nervous system and disorders of the mind in the succeeding years at the bedside. It is hardly to be expected, however, that with our already far too crowded curriculum we can hope for very great improvement in this direction. Year by year our schools find themselves obliged to cut down rather than to allocate additional time to the teaching in any one subject; and if this is true even of the fundamental subjects, what chance is there for special ones? Our needs as potential neurologists might be met, in course, only on the remote chance that the anatomist, physiologist, pathologist, physician and surgeon, as chiefs of major departments in any one school, should all happen to have leanings in our direction, for then an inter-departmental neurologic seminar might at least be held as an optional extracurricular exercise.

But, after all, possibly the best we can expect is that enough may be given to undergraduates to so far stimulate the interest of a few

of the better students that they will wish to pursue neurology as a post-graduate study. There is such a thing as specializing too early, and a thorough grounding in general subjects is perhaps even more essential for us than for any other specialists, since we more than any others tend to overlap the general field of medicine. The chief drawback lies in the fact that for the further preparation of those who have been attracted into neurology, opportunities for advanced study are far from ideal.

As I see it, the only possible solution is through the establishment of a well endowed national institute which will become the inevitable graduate school through which all ambitious students of the nervous system will wish to pass whether they are destined to become psychiatrists, neurologists, neuropathologists, neurosurgeons, or to engage in some of the sociological aspects of neurology. Given an institute free from any tendencies toward local chauvinism, with its departmental chiefs on a full-time basis engaged in research and teaching, with well equipped laboratories, with a library and bureau of publication, and with small hospital units for psychiatry, for neurology and for neurosurgery, there could arise a real American school of neurology. And from such a central institute there would radiate throughout the country such influences as would put the subject in which we are mutually interested on an entirely different plane. Such a foundation as this, which we could all support, would effectually check our tendency to drift apart into minor, somewhat combative, and unstable groups with ill-defined boundaries; psychopathologists, neurosurgeons, and so on, each speaking a language somewhat unintelligible to the others and with the consequent loss of a proper sense of that interdependence in which strength can only lie. On all this I have harped on a former occasion when a few of us, members of this Association, who had been thrown together in the war had vain hopes that something of the sort might come to pass in our generation.<sup>6</sup> But come it will, some day, I am confident; and that surgery will be an essential part of any such program is reasonably assured. Meanwhile we must somehow provide temporary training-grounds for those with surgical capabilities who would enter this field, for neurology cannot expect the spontaneous generation of many Horsleys. He, alas, was an isolated figure without surgical progeny, and men of his type are today greatly needed in many an institution here and abroad where the neurosurgeon as yet has not entered.

Traditionally the surgeon is cut from a different piece of cloth than the physician and, because of the time-consuming and fatiguing nature

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6. Concerning the Establishment of a National Institute of Neurology, *Am. J. Insan.* 76:113 (Oct.) 1919.

of the manual work he is called on to perform, his intellectual attitude toward his activities tends to be on a lower plane than the physician's: so at least they give us to understand. Admittedly such an attitude is likely to be held by those who either do their work to order or who largely limit themselves to the cut-and-dried procedures of an established sort, which require little preliminary study and have a fairly certain outcome. But in a new and difficult field like ours this is an impossible attitude if we are to make any significant advances and are to hold the confidence of our fellows. Indeed, a thorough grounding in neurology, not mere operative dexterity, should be the *sine qua non* for membership in this body; and this, I like to think, means nothing more than that as neurologists some of us are doing our own surgery rather than that we are primarily surgeons who are forcing our way into the bailiwick of others.

There is no back-door to neurology, and it is expecting a great deal of the future neurologic surgeon, I am aware, but not too much, that he should have not only a good fundamental knowledge of general medicine, but an equally good foundation in general surgery. In addition, he should have acquired a working knowledge of clinical neurology as well as of the disorders of the mind and of neuropathology; indeed, I think he should make his own postmortem examinations, for nothing is likely to be more informing, and there is a tendency for both physicians and surgeons in these days of chemistry and physiology to frequent the dead-house far less than of old.

Furthermore he should be familiar with ophthalmoscopy and perimetry, as well as with the otologic and rhinologic conditions whose complications he is likely to encounter. On top of all this, he should have mastered the particularly difficult technic essential to the successful outcome of the more serious operations on the central nervous system.

When men thus qualified are finally produced there will be no possibility of a reversion to the conditions existing within the memory of many of us—conditions which the "case history" herein recorded has served to recall; when it was the dictum that "the surgeon should be invited to a consultation in a case of brain tumor only after a three months' period of medical treatment has proven unavailing." Yet these or comparable conditions are ones which in most localities still prevail. Not many physicians are willing to call on a surgeon to operate, let us say, for an abdominal complaint, unless they feel that he shares with them a thorough knowledge of the disease. Neurologists have been slow in adopting this principle of action.

Whatever his specialty may happen to be, it is only when a surgeon is shouldered with the responsibility of acting largely on his own diag-

noses that he will be impelled seriously to study his own cases before they come to the operating table and will be inclined to follow the results of his procedures to the end to see wherein his mistakes can be rectified on subsequent occasions. On no other basis will he be likely to see all round his subject; on no other basis will he be likely to contribute anything to it by carrying his problems to the laboratory; on no other basis will he set a safe example for his pupils to follow.

## STUDIES IN DECEREBRATION

### I. A METHOD OF DECEREBRATION\*

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Associate Professor of Neurology, Northwestern University  
Medical School

AND

LOYAL E. DAVIS, M.D., M.S., PH.D.

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CHICAGO

Decerebrate animals have been produced chiefly by ablation of the cerebrum and interbrain or transection of the brain stem.

These mutilating operations are attended by a high mortality and associated with a great degree of shock. The exact level of decerebration cannot be predetermined and complete decerebration is not assured until postmortem examination. Frequently some part of the brain cephalad to the decerebration remains intact. Hemorrhage is uncontrolled and the degree of hemorrhage immeasurable. The shock obscures the picture and the time of its disappearance cannot be accurately determined. An animal decerebrated by such methods shows symptoms which result not only because of the operative removal of function of certain parts of the brain, but also because of compression by hemorrhage, shock and mutilation of an immeasurable area of tissue adjacent to the operative wound.

Other methods have been employed to destroy certain parts of the nervous system; for example, cocainization and freezing. Spinal animals have been successfully produced by the injection of foreign substances, such as chloroform, into the vertebral or carotid arteries.

Hill<sup>1</sup> has shown that occlusion of the blood supply to the central nervous system for fifteen minutes produces damage from which there can be no recovery. This has been corroborated by many workers and Stewart, Guthrie, Burns and Pike<sup>2</sup> produced excellent spinal animals by combined occlusion of the vertebral and carotid arteries, ligating the right innominate and the left subclavian arteries. Subsequent injec-

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\* Exhibited at the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, May 31, 1923.

1. Hill, Leonard: *Physiology and Pathology of the Cerebral Circulation*, London: J. and A. Churchill, 1896.

2. Stewart, G. M., Guthrie, V. C., Burns, R. L., and Pike, F.: *J. Exper. Med.* 8:289, 1906.

tions of indigo-carmin showed no dye above the level of the calamus scriptorius and usually no higher than the third or fourth cervical segment of the spinal cord.

It follows that if a method could be devised whereby complete anemia of a certain isolated part of the cerebrum could be produced for a period of time longer than fifteen minutes, that part of the brain would be rendered functionless.

Studies in comparative anatomy have shown that the cerebral arteries are laid down with remarkable precision and the distribution is closely associated with the function of the part supplied. According to Stopford,<sup>3</sup> who studied the distribution of blood vessels to the pons and medulla by means of injection experiments, the nutrient vessels to these parts are true end arteries and may be utilized in the investigation of function of certain parts of the medulla and pons.

It is possible, then, in the absence of anastomosis, to interrupt the continuity of the blood stream in the basilar artery by ligature and thus to separate the part caudad from that cephalad to the ligature. If the carotid arteries are then tied, complete anemia of the brain cephalad to the ligature on the basilar artery is produced.

#### ANATOMY OF THE BASILAR CIRCULATION IN THE CAT

The vertebral arteries of the cat, after giving off a large branch which passes laterad and dorsad to anastomose in the muscles of the neck with a branch of the occipital artery, unite at the anterior border of the foramen magnum to form the basilar artery. The basilar artery passes cranialward along the ventral median line of the brain stem. Just before their union to form the basilar artery the vertebrals each give off a small branch which passes caudad and mediad. These branches unite in the median line to form the anterior spinal artery which passes caudad the entire length of the spinal cord. (Fig. 1).

At the caudal border of the pons the basilar artery gives off a large branch on each side, the posterior inferior cerebellar artery. These vessels ramify on the surface of the inferior half of the cerebellum. At the cranial margin of the pons the posterior superior cerebellar arteries arise from the basilar artery and crossing the cerebral peduncles supply the superior half of the cerebellum. Just cranial to the origin of these vessels, the posterior cerebral arteries arise and pass to the caudal portion of the cerebrum. The basilar then divides and passes cranial at the side of the hypophysis to join the posterior communicating branches from the carotid plexus. A large vessel arising from the carotid plexus of the internal maxillary artery, passes into the cranial cavity through the orbital fissure and

3. Stopford, J. S. B.: *J. Anat. & Physiol.* 1:131 and 255, 1916.

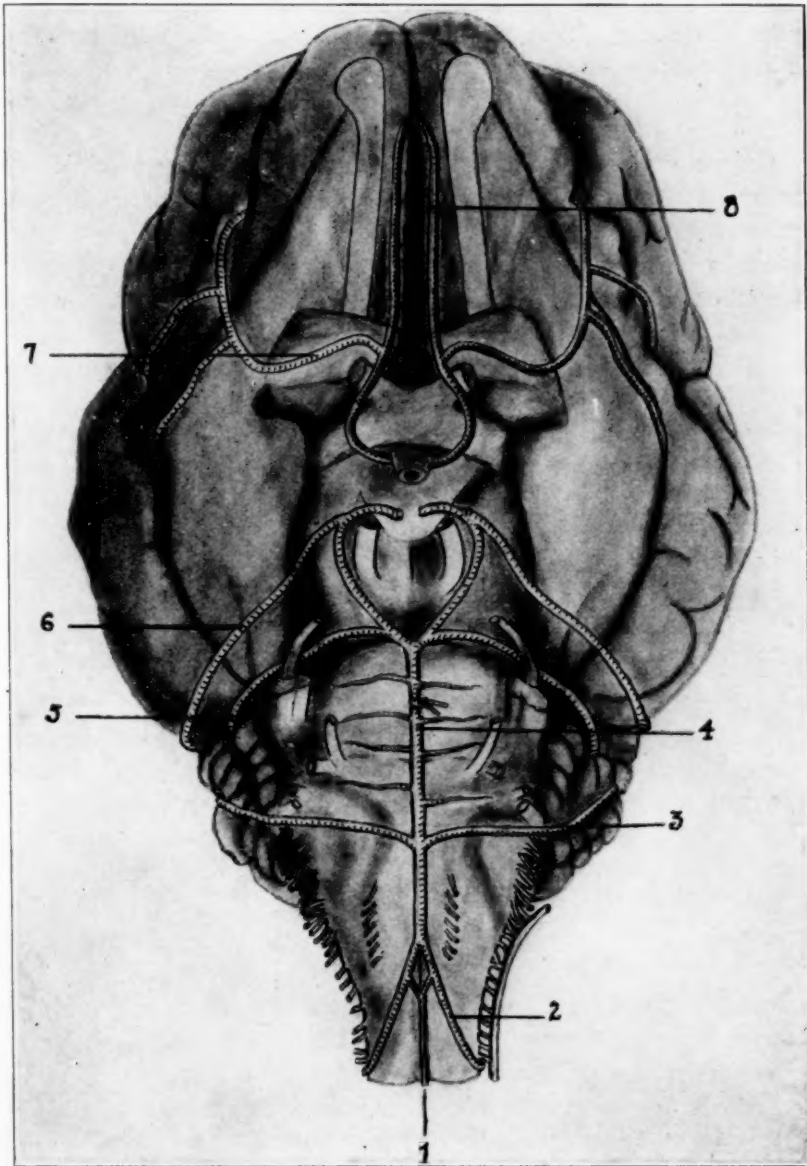


Fig. 1.—Drawing of the basal surface of the cat's brain, showing the arrangement of the vessels constituting the circle of Willis. 1. Anterior spinal artery; 2. vertebral artery; 3. posterior inferior cerebellar artery; 4. basilar artery; 5. posterior superior cerebellar artery; 6. posterior cerebral artery; 7. middle cerebral artery; 8. anterior cerebral artery.

lies within the skull at the side of the hypophysis. It gives off the posterior communicating branches, the middle cerebral and anterior cerebral arteries. These vessels form the circle of Willis and with the basilar constitute the basilar circulation.

#### METHOD OF DECEREBRATION

The method of decerebration by anemia consists of two steps: first, the ligation of the basilar artery at any desired point and, second, the ligation of the carotid arteries.

Cats were used throughout as experimental animals of choice. After they were anesthetized with ether in the usual manner they were tied to an operating table with the ventral aspects upwards. A small catheter was introduced into the trachea and connected with an ether bottle through which a stream of air was directed. The ether bottle was so arranged with valves that any desired stage of anesthesia could be secured and maintained.

A mouth gag was then fixed in place and the jaws were widely separated. This also served as a means of securely fixing the animal's head in position. A ligature having been passed through the tip of the tongue and that organ being held outside the mouth, a median line incision was made in the soft palate from the posterior edge of the hard palate posteriorly. The mucous membrane and muscles were separated from the base of the skull posteriorly so as to expose the anterior border of the foramen magnum and laterally to expose the tympanic bullae. The two flaps of mucous membrane and muscles thus made were retracted by ligatures. It is very important to obtain a field clear from tags of mucous membrane and muscle, and to expose the tympanic bullae. These latter bony prominences serve as valuable points of localization in the later operative steps. Opposite these structures one reaches a level in the pons at about the exit of the fifth cranial nerve. (Fig. 2).

A motor driven dental burr was used to trephine through the base of the skull (ventral surface of the body of the sphenoid, basisphenoid and basioccipital bones) and to expose the dura mater covering the pons and the basilar artery with its branches. This method of trephining was found to be satisfactory since it tended to stop any bleeding from the edges of the bone. This procedure was continued until a membrane-like layer of bone remained. This was then carefully removed by the use of a fine spatula. The basilar artery was then visible lying beneath the dura mater. A dural hook was used to open the dura mater and allow the escape of cerebrospinal fluid. With this exposure the basilar artery can be seen in the midline of the pons and its side branches are easily distinguishable.

If one wishes to ligate the basilar at the level of the exit of the fifth cranial nerve from the pons, the trephine opening in the skull must be made exactly opposite the middle of the tympanic bullae. If it is desired to place the ligature on the caudal end of the basilar at its formation by the two vertebrals, it is necessary to remove the anterior

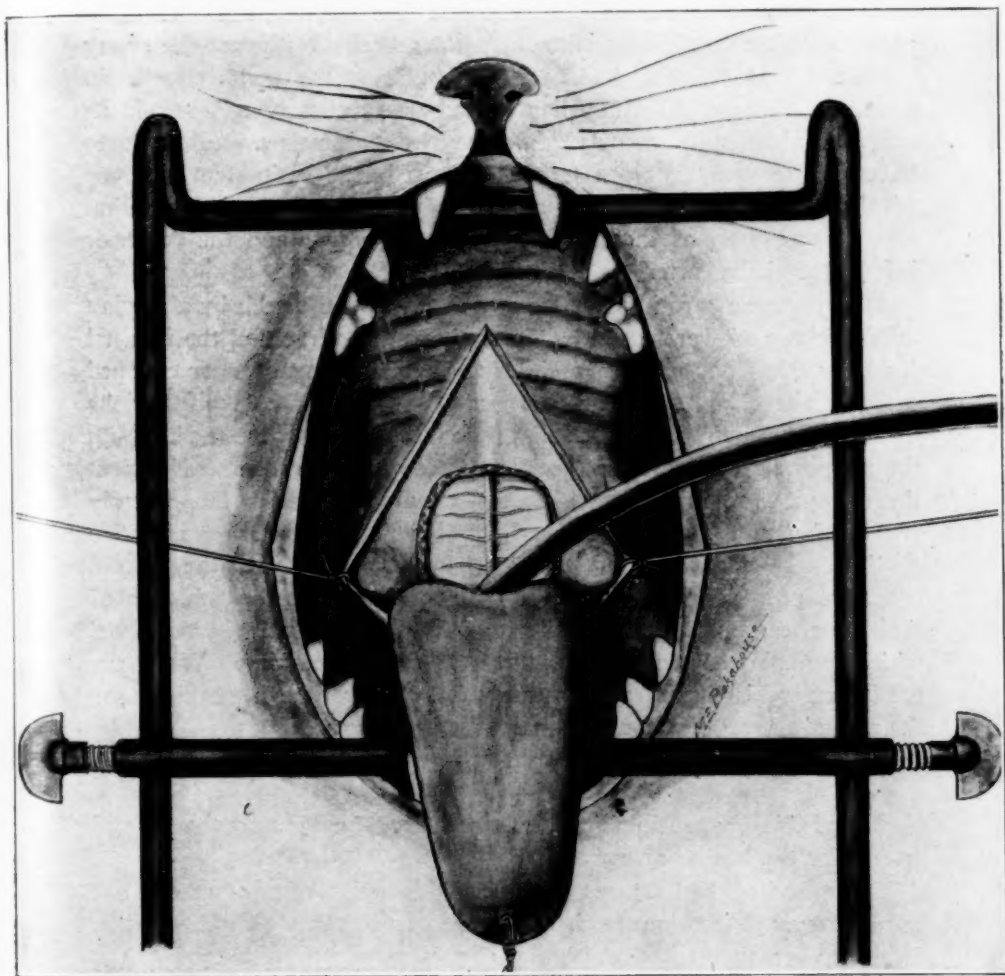


Fig. 2.—Drawing of the operative field in ligating the basilar artery showing the mucous membrane retracted to expose the tympanic bullae, and the trephine opening in the bone with the underlying basilar artery.

border of the foramen magnum before opening the dura mater. The posterior inferior cerebellar arteries arise from the basilar at a point about midway between the posterior margin of the tympanic bullae and the anterior border of the foramen magnum. The posterior superior cerebellar arteries from the basilar are just caudad to the

exit of the oculomotor nerves from the brain stem. Thoroughly to expose these vessels it is necessary to remove the dorsum sellae. This gives one access to the posterior cerebral arteries also. For such an exposure the trephine opening must be made about 0.5 cm. craniad to the anterior edge of the tympanic bullae.

After the basilar artery has been exposed at the level desired, a strabismus hook, into the end of which a fine hole has been drilled, is used to place a fine silk ligature about the vessel. With care the artery can be ligated without the slightest trauma to the underlying brain stem. We have successfully ligated this artery at varying levels in its course in about sixty animals. In some of the animals several ligatures have been applied, thus producing segments in the brain stem deprived of their blood supply. It is also possible to ligature the vertebral arteries separately as they unite to form the basilar and the posterior inferior cerebellar arteries.

The common carotid arteries were exposed in the neck by a median line incision and were separated from the vagus nerve and the internal jugular vein. They were then either tied with silk ligatures or clamped with a Crile clamp. For the purpose of studying the manifestations of nervous tissue recovering from anemia, the clamps were used. With the application of the clamps a decerebrate preparation resulted; when the clamps were removed within ten minutes, the animal apparently recovered normal function. When time is a factor it may be advisable to ligate the carotid arteries first. The recovery from the carotid operative procedure is very rapid and no change in the behavior of the animal results. The basilar artery may then be ligated in an animal which is apparently in a condition of normal functioning. When the basilar artery is ligated first, a somewhat longer time is required for the animal to recover from the operative procedure.

#### RESULTS OBSERVED

No visible change in function is produced by ligating the carotid arteries or the basilar artery alone.

Shortly after the ligature of both carotids and the basilar artery, the animal assumed the characteristic posture of decerebrate rigidity. The forelegs were thrust backward, the elbows were rigidly extended, the hindlegs were usually not as completely involved in the rigidity as were the forelegs. The tail was invariably curved stiffly upward. The head was lifted and retracted on the neck. The mouth was tightly closed. If the legs, tail or head were moved from the attitude assumed they would immediately spring back into the position of extensor rigidity. While this posture would be assumed with the animals lying on the side, the suspended position and fixed supine position favored the early production of rigidity.

A detailed description of the decerebrate preparation will be dealt with in a following communication. It may be said that the corneal reflex is preserved and the light reflex is absent. These animals without exception exhibited vibrissae, sneeze, pinna and scratch reflexes. The Magnus-deKleijn reflexes produced by manipulation of the head were likewise obtainable. Respiration was disturbed for a very brief time following the ligation and after that no measures were necessary for its maintenance. We have made no endeavor thus far to keep the animals alive for any length of time by careful nursing methods. In spite of this such animals commonly lived twenty-four hours.

The level of the section obtained by the production of anemia may be accurately determined by the peripheral injection of vital stains

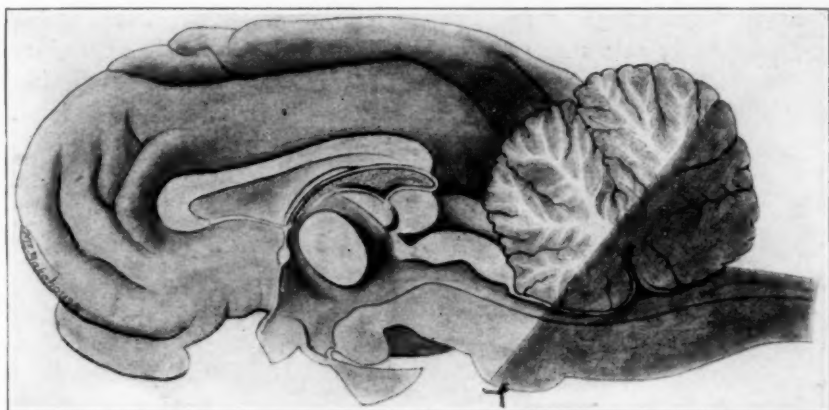


Fig. 3.—Drawing of the medial aspect of the cat's brain showing the basilar artery ligated and the extent of the vital stain over the inferior half of the cerebellum, the caudal half of the pons, the medulla and the spinal cord.

intravenously. A definite differentiation is produced between the normal and anemic part of the brain, the latter remaining colorless. Figure 3 shows the level commonly obtained by the intravenous injection of methylene blue when the basilar artery is ligated at the level of the fifth cranial nerve nuclei. It will be seen that the superior half of the cerebellum remains unstained. This preparation, therefore, in addition to being decerebrate, has lost the function of the superior part of the cerebellum. If the basilar artery is tied at the junction of the vertebrals, the whole cerebellum is rendered anemic. Whether half or all of the cerebellum was excluded from the circulation, the decerebrate rigidity seemed in no wise influenced. It would be possible to produce a decerebrate preparation leaving the cerebellum intact. The

operative difficulty, because of hemorrhage from the cavernous sinus, is such that for our purposes we have not persisted in efforts to produce such an animal. (Fig. 3).

#### COMMENT

Anemia of the central nervous system prolonged for more than fifteen minutes produces irrecoverable loss of function of the anemic portion. By means of combined ligation of the carotid arteries and the basilar artery loss of function occurs in that part of the brain cephalad to the ligature on the basilar artery. Varying levels of decerebration are therefore possible. The animal exhibits all the phenomena of decerebrate rigidity. The same result ensues whether the function of part or all of the cerebellum is abolished. Ligation of the basilar artery causes no shock and a study of the preparation may be made immediately after the operation. The result is not obscured by the effect of hemorrhage or mutilation of adjacent parts. There should be no mortality as a result of the operative procedure. The constancy of the preparations produced will permit of more accurate descriptions of the resulting phenomena and of less controversial interpretations.

## RESEARCHES ON THE PUPILLARY REACTIONS IN EPIDEMIC ENCEPHALITIS \*

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In the diagnosis of epidemic encephalitis, the ocular findings are of primary importance. Pupillary changes are receiving increasing attention in the last two years and frequent references have appeared in literature concerning the occurrence of the Argyll Robertson pupil. This sign, which formerly has been considered pathognomonic for neurosyphilis, has lately been reported in chronic alcoholism, injuries, and tumor of the midbrain. In spite of its occasional occurrence in these conditions, its frequent presence in a condition as prevalent as epidemic encephalitis would tend to lessen its diagnostic value to clinicians.

In reviewing the pupillary reports in the literature, very frequent mention is found of absent or sluggish light reflexes. There is a surprisingly small number of cases in which a true Argyll Robertson sign was brought out. Among the authors who record cases which they feel certain exhibit an Argyll Robertson sign are Bonhoeffer,<sup>1</sup> Economo,<sup>2</sup> Pette,<sup>3</sup> Dreyfus,<sup>4</sup> Nonne,<sup>5</sup> and Wilson.<sup>6</sup> Waardenburg<sup>7</sup> mentions "Transitory Argyll Robertson phenomena"; Adler<sup>8</sup> concludes that the Argyll Robertson pupil in encephalitis should be diagnosed with the greatest caution. Other cases, such as that of Dickinson,<sup>9</sup>

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1. Bonhoeffer, K.: *Neurol. Centralbl.* **39**:286, 1920; *Deutsch. med. Wchnschr.* **47**:229, 1921.

2. Economo: *Wien. med. Wchnschr.* **71**:1322, 1921.

3. Pette: *Deutsch. Ztschr. f. Nervenheilk.* **76**:32, 1923.

4. Dreyfus, G. L.: *München. med. Wchnschr.* **67**:538 (May 7) 1920.

5. Nonne: *Deutsch. Ztschr. f. Nervenheilk.* **64**:185, 1919, and **65**:220, 1920.

6. Wilson, S. A. K.: *J. Neurol. & Psychopath.* **2**:1 (May) 1921.

7. Waardenburg, P. J.: *Am. J. Ophthalmol.* **4**:580 (Aug.) 1921.

8. Adler, E.: *Med. Klin.* **17**:13 (Jan. 2) 1921.

9. Dickinson, G.: *Am. J. Ophthalmol.* **3**:587 (Aug.) 1920.

cannot be included because of a lack of some essential point in the diagnosis, that of Junius<sup>10</sup> being of little value because of the presence of a positive blood Wassermann reaction. This variety of opinion may be explained on several grounds, the most important of which is the difficulty of securing the necessary data in an acute case. The convergence reaction can only be elicited with the full cooperation of the patient; any doubt on the point must greatly confuse the diagnosis. Again, the frequently changing findings in the same patient, ranging from normal to iridoplegia during the course of the disease, make a final decision even more difficult. It seemed worth while to investigate a series of epidemic encephalitis cases with such a psychic state that they would cooperate in the more delicate diagnostic procedures. This was only possible in the subacute and chronic cases.

Before proceeding to the examination of the material, it would be well to define our understanding of the Argyll Robertson sign. Argyll Robertson<sup>11</sup> himself defined it as follows: "The pupil was insensible to light, but contracted still further during the act of accommodation for near objects." He uses the phrase "during the act of accommodation for a near object" in the sense of focusing the eyes for a near object. He was therefore testing the reaction which we ordinarily designate the convergence-accommodation reaction of the pupil. He adds in the same paper that the patient "with either eye, was able to read fine print." This implies, although it does not specifically state, that the patient's accommodative power (ciliary muscle) was intact.

Some of the modern textbooks, including that of Purves-Stewart,<sup>12</sup> hold to the original definition: "There is a loss of the light reflex, with a preservation of the reaction to accommodation and convergence." Others, with Jelliffe and White,<sup>13</sup> would allow a "partially lost" light reflex, or, with Kinnier Wilson,<sup>6</sup> an "obvious diminution" of the light reflex, to be included in our present conception of the sign. Other observers have included an involvement of the accommodative power of the ciliary muscle in the definition of the Argyll Robertson sign. Some cases of Argyll Robertson pupils noted as having occurred in encephalitis, including the often quoted case of Nonne,<sup>5</sup> show paresis of the accommodative power of the ciliary muscle. We have not, however, considered that these cases could be properly included. It seemed to us an unwarranted extension of the original definition to

10. Junius: *Ztschr. f. Augenheilk.* **44**:46, 1920.

11. Robertson, Argyll: *Edinburgh M. J.* **15**:487.

12. Stewart, Purves: *The Diagnosis of Nervous Diseases*, Ed. 5, New York: E. B. Treat & Co., 1922, p. 126.

13. Jelliffe, S. E., and White, W. A.: *Diseases of the Nervous System*, Ed. 4, Philadelphia: Lea & Febiger, 1923, p. 34.

include them among those exhibiting the Argyll Robertson sign. We have classed them under the caption of *ophthalmoplegia interna*.

The extension of the limits of the definition of the Argyll Robertson pupil to include the incomplete or incipient forms, while advantageous from one point of view, tends to introduce inaccurate and obscure terms such as "sluggish," "questionable" and "doubtful," pupillary reaction. In order to minimize the inaccuracies due to the personal equation in the study of the pupillary reaction to light, we availed ourselves of the pupilloscope.

#### METHODS OF EXAMINATION

In testing the material we used the following methods:

(A) *The Light Reaction*.—We examined for the the light reaction both by the ordinary methods and by the pupilloscope. In this instrument, designed by von Hess, the patient's eye is observed through a telescope of eight magnifications. From a constant source of illumination a beam of light is cast upon the eye. This beam of light is made to traverse a frame which consists of two compartments (Fig. 1). The upper compartment contains a gray glass of known permeability to light. The lower contains two gray prisms which are calibrated and can be superimposed one on the other to any desired extent by means of a micrometer screw. The amount of light passing through the lower compartment is varied by changing the position of the prism. The frame is then swung up and down so that the beam of light passes successively through the upper and the lower compartments. When the permeability of the compartments is equal we are illuminating the eye with a constant beam of light. By changing the permeability of the lower compartment it is in our power to illuminate the eye with any desired difference of light. We now determine the least difference of light which just suffices to elicit reaction of the pupil and this difference of light is a numerical index of the sensibility of the pupil, and, therefore, of the pupillary reflex arc. The prisms are most permeable to light at their apices and least at their bases so that the permeability of the systems of prisms varies according to the positions of the superimposed portions. The position of the prisms and the amount of light passing through are shown on the micrometer scale and we can thus measure accurately the amount of light necessary to secure reactivity of the pupil (Fig. 2). The smallest difference of light intensities between which the normal pupil can distinguish is as 95 to 100. The value is constant, is irrespective of the age of the individual, and is independent of the state of adaptation. In our table we have appended the scale readings as found with the particular gray glasses used in this series: glass I, in which differences of five are within normal limits; with glass IV, differences of two are within normal limits. With the

aid of the pupilloscope, about 20 per cent. of the cases showing doubtful pupillary reactions were definitely shown to be either pathologic or within normal limits.

(B) *Convergence-Accommodation Reaction.*—The ordinary test method consists of making the patient look into the distance and then look fixedly at a near object, preferably his finger. Of the sources of

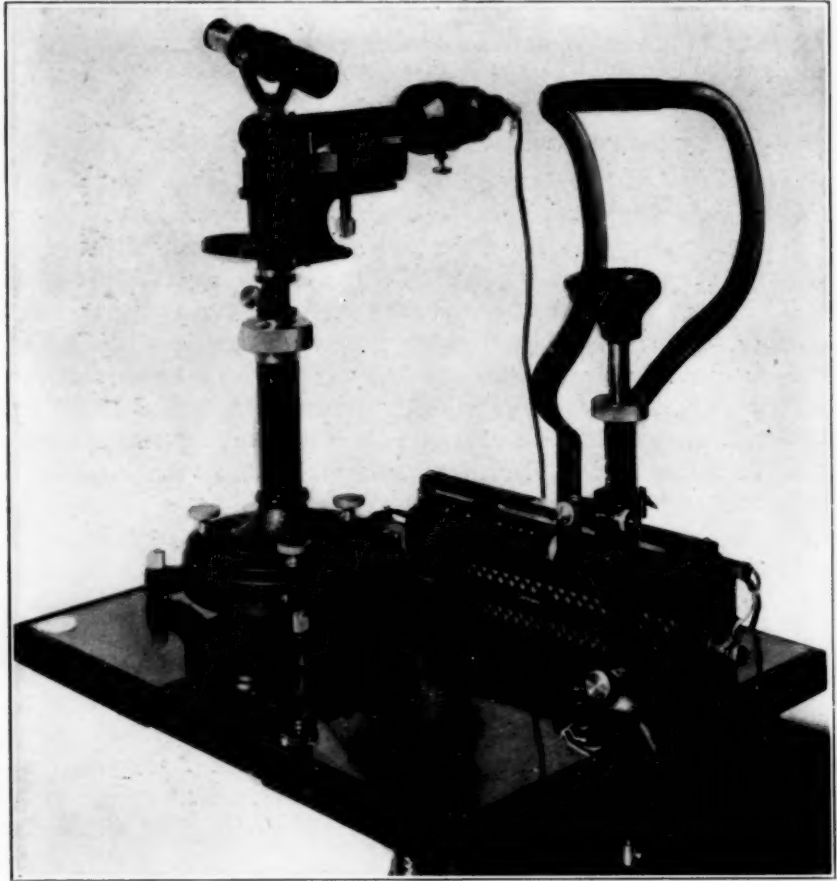


Fig. 1.—The pupilloscope.

error, apart from difficulties in determining the quantitative reaction of the pupil, the most constant is that resulting from a sluggish psychic state. The examiner is frequently in doubt as to how much effort the patient is making. Moreover, no quantitative measurement has thus far been elaborated for the determination of the convergence reaction, yet that is one of the most commonly used neurologic tests and one on which considerable reliance is placed.

In view of the above mentioned sources of error and of the fact that it is only an associated act in focusing for near objects, the essential act being the convergence of the eye balls and the accommodative power of the ciliary muscle, it would seem that the clinical usefulness of the convergence reaction is much overrated.

Seldom, on the other hand, is the accommodative power tested in a neurologic examination. We tested for it at first only in the way of

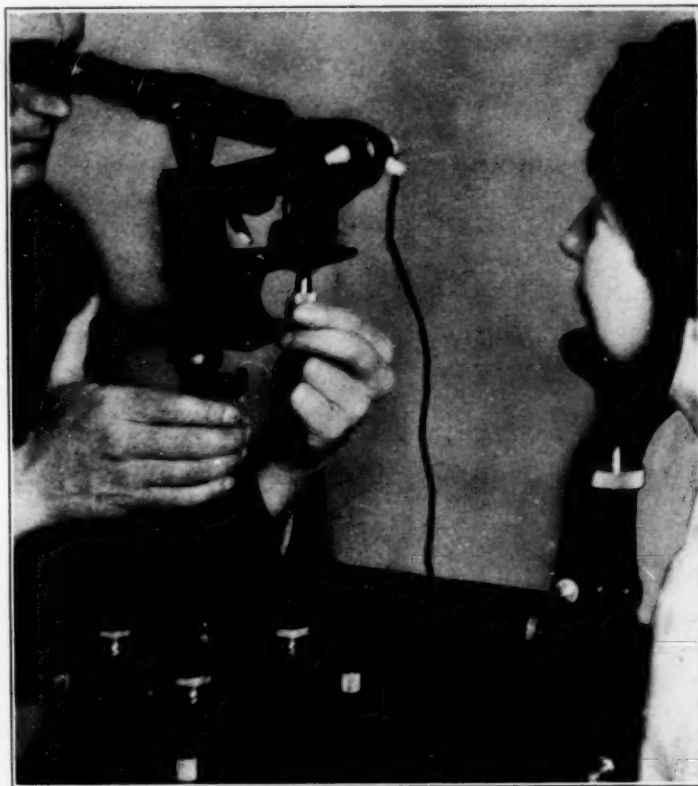


Fig. 2.—Showing how the operator, changing the position of the prism, regulates the amount of light passing through the lower compartment.

routine, especially as it could be measured accurately. We soon found that the accurate measurement of the accommodative power furnished most important data.

*Accommodative Power.*—The accommodative power is an expression of the power of the ciliary muscle. This can be measured by approximating fine print to the emmetropic eye until the type just begins to blur. This distance increases from youth to old age with



Fig. 3.—Testing the accommodative power.

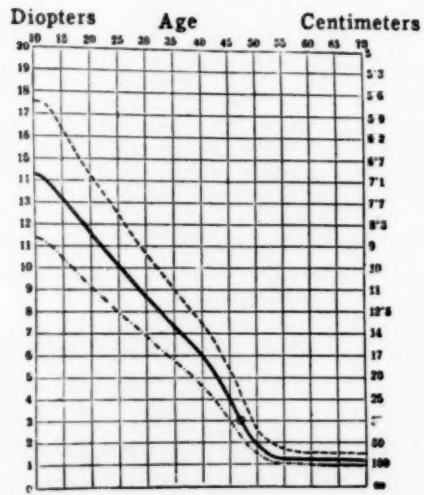


Fig. 4.—Range of accommodation at different ages; after Donders, amended by Duane, Fuchs' Textbook of Ophthalmology.

but very slight variations. The accommodative power may be expressed in centimeters; the starting point for the measuring must, of course, be constant, namely, focus for infinity or emmetropia. In each case, therefore, the refraction of the eye must be known, or, as is more convenient, the eye be corrected for emmetropia with glasses. When this latter is the case, but only then, the neurologist can accurately measure the distance between the cornea of the eye and the print held at the position where type just begins to blur. Then, with the appended table (Fig. 4) and the knowledge of the patient's age, he can compare the accommodative power with the normal on the table for that age, and determine whether the accommodative power (ciliary muscle) is affected or not. True, many pitfalls are encountered, which only experienced ophthalmologists can properly adjudge, but for the grosser lesions of the ciliary muscle, the neurologist can obtain a very fair idea with this rough and ready method.

#### ANALYSIS OF MATERIAL

The material consisted of thirty-six cases, most of them ambulatory and all of them in such a psychic state as to be able to cooperate in the examination. The blood and spinal fluid Wassermann reactions in each case were normal. No trace of the Argyll Robertson pupil was found. It is, of course, conceivable that our material, consisting of subacute and chronic cases, might contain a less number than an acute material would have done. However, the fact that our patients could all cooperate to the fullest extent gave an opportunity to demonstrate defects in convergence which, in lethargic patients, would have been impossible. Again, the routine testing for the accommodative power frequently made the diagnosis of ophthalmoplegia interna, which if not tested for, might conceivably have been classed in an Argyll Robertson group.

The sluggish pupils found clinically or with the aid of the pupilloscope were accompanied, either by a reduced convergence reaction, or reduced accommodative power. Frequently all three were involved. In the only instance in which a sluggish light reaction was obtained by the pupilloscope in the face of a normal convergence reaction and accommodative power, the history showed a recent injection of homatropin for diagnostic purposes.

Of ophthalmoplegia interna there were sixteen cases (44 per cent.), one of which showed an ophthalmoplegia interna in one eye and an isolated paresis of accommodative power in the other. A doubtful iridoplegia occurred once—this may have been an ophthalmoplegia interna.

CHART 1.—Pupillary Findings in Thirty-Six Cases of Epidemic Encephalitis, Selected for Ability to Cooperate

Case No.	Age	Duration Disease	Wassermann		Diagnosis	Size of Pupil in Millimeters		Shape of Pupil	Light Reaction		Convergence Reaction	Accommodative Power	Conclusion
			Spinal Fluid	Blood		Right	Left		Clinical	Pupilloscope			
1	31	6 mo.	Negative	Negative	Encephalitis	5	4	Round	Sluggish	18-23 reduced	Sluggish	Reduced	Ophthalmoplegia Interna
2	59	2 yr.	Negative	Negative	Encephalitis (parkinsonian)	3.5	4	Ovoid	Absent	Absent	Sluggish	None	Iridoplegia
3	27	3 mo.	Negative	Negative	Encephalitis	3.5	2.5	Round	Normal	35-40 35-40 normal	Sluggish	Normal	Normal
4	39	2 mo.	Negative	Negative	Encephalitis	2.5	2.5	Round	Sluggish	34-42 34-42	Sluggish	Reduced	Ophthalmoplegia Interna
5	49	2 yr.	Negative	Negative	Encephalitis	5	4.5	Round	Normal	Normal	Normal	Normal	Normal
6	76	3 mo.	Negative	Negative	Encephalitis	3	2	Round	Right sluggish	37-40 37-40	Right sluggish	None	Normal
7	25	6 wk.	Negative	Negative	Encephalitis	3	3	Round	Normal	37-40 37-42 normal	Normal	Reduced	Paresis of accommodative power (isolated)
8	20	2 yr.	Negative	Negative	Encephalitis	5.5	4	Ovoid	Trace	Trace	Trace	Reduced	Ophthalmoplegia Interna
9	44	2 yr.	Negative	Negative	Encephalitis (parkinsonian)	3.5	3.5	Round	Normal	35-40 37-39 normal	Sluggish	Reduced	Paresis of accommodative power (isolated)
10	46	2-4 yr.	Negative	Negative	Encephalitis (parkinsonian)	3	3	Round	Normal	38-43 37-42 normal	Normal	Reduced	Paresis of accommodation (isolated)
11	31	3 yr.	Negative	Negative	Encephalitis	4.5	3.5	Round	Right sluggish	Right reduced	Right sluggish	Reduced	Right ophthalmoplegia interna; left paresis of accommodative power (isolated)
12	32	3.5 yr.	Negative	Negative	Encephalitis	4.5	4.5	Round	Normal	Normal	Absent	Absent	Paresis of accommodative power
13	42	1.5 yr.	Negative	Negative	Encephalitis	3.5	3.5	Round	Normal	Normal	Normal	Reduced	Paresis of accommodative power (isolated)
14	30	5 wk.	Negative	Negative	Encephalitis	4	4	Round	Sluggish (?)	Normal	Normal	Reduced	Paresis of accommodative power (total)
15	18	8 mo.	Negative	Negative	Encephalitis	5	6	Ovoid	Sluggish	23-39 19-38 reduced	Left sluggish	Reduced	Ophthalmoplegia Interna left more than right
16	26	5 wk.	Negative	Negative	Encephalitis	3.5	3.5	Round	Normal	Normal	Normal	Reduced	Paresis of accommodative power (isolated)
17	26	2 yr.	Negative	Negative	Encephalitis	3	3	Round	Normal	25-40 25-40 reduced	Absent	Reduced	Ophthalmoplegia Interna

18	16	1 mo.	Negative	Negative	Encephalitis	5.5	5	Round	Normal	25-40 reduced 36-40 35-42 normal 38-39 36-39 normal	Absent	Reduced	Normal (?)
19	18	2 yr.	Negative	Negative	Encephalitis	3.5	3.5	Round	Normal	36-40 35-42 normal	Normal	Reduced	Paresis of accommodative power (isolated)
20	36	2 mo.	Negative	Negative	Encephalitis	3.4	3.4	Round	Normal	38-39 36-39 normal	Normal	Normal	Normal
21	43	3 mo.	Negative	Negative	Encephalitis	3.4	3.4	Round	Normal	38-39 35-40 normal	Normal	Reduced	Paresis of accommodative power
22	16	2 mo.	Negative	Negative	Encephalitis	5	4	Round	Sluggish	26-40 30-43 reduced	Normal	Reduced	Ophthalmoplegia interna
23	38	3 yr.	Negative	Negative	Encephalitis	3	3	Round	Normal	Normal	Normal	Reduced	Paresis of accommodative power (isolated)
24	26	4 yr.	Negative	Negative	Encephalitis	4	4	Round	Sluggish	12-48 30-46 reduced	Reduced	Reduced	Ophthalmoplegia interna
25	53	1.5 yr.	Negative	Negative	Encephalitis	3	3	Round	Normal	38-40 38-40 normal	Normal	Absent	Paresis of accommodative power
26	38	1 yr.	Negative	Negative	Encephalitis	3	3	Round	Normal	35-40 35-40 normal	Normal	Normal	Normal
27	39	3 yr.	Negative	Negative	Encephalitis	3	3	Round	Normal	35-40 28-45 reduced	Normal	Reduced	Right, paresis of accommodative power; left, ophthalmoplegia interna
28	23	2 yr.	Negative	Negative	Encephalitis	3	2.5	Ovoid	Sluggish	32-48 20-50 reduced	Sluggish	Reduced	Ophthalmoplegia interna
29	26	2 yr.	Negative	Negative	Encephalitis	3.5	3.5	Round	Normal	Normal	Normal	Normal	Normal
30	22	2 mo.	Negative	Negative	Encephalitis	5	7	Ovoid	Sluggish	30-45 reduced	Sluggish	Reduced	Ophthalmoplegia interna
31	37	4 yr.	Negative	Negative	Encephalitis	3	3	Round	.....	40-42 40-42 normal	Normal	Reduced	Paresis of accommodation (isolated)
32	17	2 yr.	Negative	Negative	Encephalitis	3	3.5	Round	.....	33-52 39.5-40.5 reduced	Reduced	Reduced	Ophthalmoplegia interna
33	26	1 yr.	Negative	Negative	Encephalitis	3.5	3.5	Round	.....	36-46 36-44 reduced	Sluggish	Reduced	Ophthalmoplegia interna
34	36	2 mo.	Negative	Negative	Encephalitis	3	3	Ovoid	.....	30-44 38-44 reduced	Normal	Reduced	Ophthalmoplegia interna
35	27	1 yr.	Negative	Negative	Encephalitis	3.5	3.5	Round	Sluggish	Reduced	Sluggish	Reduced	Ophthalmoplegia interna
36	28	6 mo.	Negative	Negative	Encephalitis	3	3	Round	Normal	Reduced	Normal	Reduced	Ophthalmoplegia interna

Reduction of the accommodative power occurred as an isolated sign in thirteen cases (36 per cent.), in addition to being present in the sixteen cases of ophthalmoplegia interna (44 per cent.). Thus we found the accommodative power affected in 80 per cent. of all cases, making the paresis of the ciliary muscle the outstanding ocular lesion in epidemic encephalitis.

Six cases showed no abnormality.

The size of the pupils averaged from medium to large. The excursion of the pupil, even when it reacted to light, was small. In shape, thirty pupils were round and six ovoid. This would seem to show that the really irregular pupil is rare in encephalitis.

Comparing our findings in this series of encephalitis with a like series composed of cerebrospinal syphilitic cases, it is interesting to note that the Argyll Robertson pupil was found to be present in 68

CHART 2.—Comparative Pupillary Findings in Encephalitis and Syphilis

Light Reaction	Convergence Reaction	Accommodative Power	Diagnosis	Encephalitis, No. Cases	Per Cent.	Syphilis, No. Cases	Per Cent.
Diminished	Normal	Normal	Argyll Robertson.....	0	0	52	68
Diminished	Diminished	Diminished	Ophthalmoplegia interna	16	44	3	3.9
Diminished	Diminished	Normal	Iridoplegia.....	1?	2.7?	7	9.2
Diminished	Normal	Diminished	Reduced A-P (Paresis of Accommodative Power)	13	36	1	1.3
Normal			Normal.....	6	16	13	17

per cent. of the latter cases, ophthalmoplegia interna in only 3.9 per cent. Iridoplegia was found in 9.2 per cent., compared with 1 per cent. in encephalitis. Only one case was found exhibiting an isolated reduction of the accommodative power.

Other pupillary findings were: the prevalence of anisokoria; the absence of an irregularity of the pupil, the variation from the normal being the ovoid, the rule, the round pupil. Mydriasis was the rule, although we did have some small pupils.

#### CONCLUSIONS

1. No Argyll Robertson pupils were found in thirty-six selected cases of epidemic encephalitis.
2. A sluggish light reaction was always part of an ophthalmoplegia interna.
3. Isolated paresis of accommodation, in which the pupillary reaction was normal, was also a frequent sign.
4. The pupil in encephalitis is often mydriatic and is round or ovoid, in marked contrast to the serrated irregularity of the syphilitic pupil.

5. As the result of our investigations we would define an Argyll Robertson pupil as one in which the light reaction is reduced or absent in the presence of a normal convergence-accommodation reaction, in agreement with the definition of Argyll Robertson himself, but we believe it necessary to stipulate also (as he seems to have implied) that the accommodative power (ciliary muscle) be intact. Unless this be done an ophthalmoplegia interna may be erroneously diagnosed as an Argyll Robertson sign.

#### DISCUSSION

DR. FRANCIS X. DERCUM, Philadelphia: While I have made no studies with the pupilloscope, the authors' findings are in accord with my own experience, that absence or disturbance of the light reaction are rarely found in encephalitis. On the other hand, in a large number of cases we find impairment of the reactions in convergence and accommodation. I believe this sign to be of the utmost diagnostic value. Not infrequently the common symptom of diplopia is absent, but in doubtful cases I have always felt that if accommodation is impaired ever so little, one should favor the diagnosis of encephalitis. Furthermore, paresis or paralysis of the ciliary muscle in my experience is one of the earliest symptoms, occurring as early as the diplopia.

DR. FOSTER KENNEDY, New York: We have been misleading students by teaching that the Argyll Robertson pupil is a sign of syphilis only. It can occur from toxic, infective or mechanical attack on the oculomotor nuclei. It frequently occurs in epidemic encephalitis and as an instance of its mechanical origin, I have mentioned a case of tumor growing on the inner side of the right temporosphenoidal lobe, producing pressure on the midbrain. This pressure was gradual and, at first, not continuous. There were passing periods in which there was loss of conjugate movement upward of the eyes. Such periods might last but a few hours. When there was loss of conjugate movement upward, the Argyll Robertson pupil was present; the phenomenon disappeared with the removal of the pressure on the quadrigeminal region. Later Dr. Elsberg operated on this patient. The location of the lesion and the mechanics of the situation as I have described it were proved later by necropsy. It is the only instance I know, of a purely mechanically produced Argyll Robertson pupil, and is consequently important.

DR. ISRAEL STRAUSS, New York: During the epidemic of encephalitis we did not find the Argyll Robertson pupil frequently, but often found a pupil fixed both to light and in accommodation; it is an interesting fact that the fixed pupil persisted for a long time. In some cases the only symptom persisting for some months is the fixed pupil, but in many cases it is the difficulty in accommodation which interferes with reading. Involvement of accommodation and of the musculature of the lens is present early. Frequently diplopia is not complained of at the beginning, but rather indistinctness of vision which persists. An ophthalmologist in the hospital claims that with the pupilloscope he is able to diagnose an Argyll Robertson pupil before the neurologists. In one patient he asked me to discern which eye showed interference with the light reflex or accommodation reflex. He thought that it could not be done. I noted the eye in which there was the very slightest defect in reaction, which he had not perceived. The pupilloscope is an instrument of great precision and one which may in the future have a great practical application. We have not used it in cases of epidemic encephalitis.

DR. DONALD J. MACPHERSON, Boston: In fifty-four cases of acute encephalitis that have been in the Brigham Hospital since 1919, sixteen showed failure of convergence. Of these there were four in which no contraction of the pupils to light could be demonstrated. It was also noted that in three of these cases in which the light reaction was not impaired, there was no reaction in accommodation.

DR. S. A. KINNIER WILSON, London: I have seen more than one typical case of epidemic encephalitis with a characteristic Argyll Robertson phenomenon. It occurs also in disseminated sclerosis without doubt, and there are a number of cases which are certainly not syphilitic in which one can get a characteristic Argyll Robertson pupil. It occurs in conditions in which there is neoplastic change in the vicinity of the aqueduct. All that is necessary is that the lesion should be in a certain place. It does not matter what the pathologic nature of the lesion is. The only difficulty is that we get it much more commonly in syphilis than we do in anything else, because there is a specific action in connection with the syphilitic virus. It has a sensory affinity. In the case of the light reflex we have to suppose that there is a special affinity of the syphilitic virus for the central protoneuronic terminations of the visual apparatus, and if they are involved, just as the spinal dorsal roots are involved, there will be interference with the light reflex. We get a paralysis of the light reflex without paralysis of the accommodation reflex. In encephalitis lethargica, one may also observe the reverse of the Argyll Robertson phenomenon, which is readily enough explained.

DR. ALFRED GORDON, Philadelphia: Nonne, Economo and Naeff observed a large number of encephalitic cases during the epidemic in Munich, and found the Argyll Robertson pupil in this affection. I did not use the accurate instrument of which Dr. Mehrtens spoke. The Argyll Robertson pupil rapidly disappears in lethargic encephalitis, but is persistent in syphilitic encephalitis.

DR. HENRY C. MEHRTENS, in closing: Dr. MacPherson's series was very much to the point from the standpoint of the cases that we investigated, particularly those patients in whom he found absent or sluggish light reactions with sluggish convergence-accommodation reaction. According to the ordinary terminology the cases would be classed as iridoplegia, which the results of our series would indicate is suggestive of syphilis in which it occurs much more frequently than in encephalitis. In Dr. MacPherson's cases it would have been most interesting to measure the accommodative power of the ciliary muscle; in syphilis this is rarely involved while in encephalitis it is frequently involved. This brings out again the importance of testing the accommodative power of the ciliary muscle in all suspected cases of Argyll Robertson pupils.

There is no reason why an Argyll Robertson could not occur with any lesion placed in the exact position necessary to produce the sign. Again we would say, however, that the cases in which we finally determine an Argyll Robertson sign to be present should have the accommodative power tested in addition to the light and convergence reaction, otherwise we may be adding to our series a considerable number of examples of ophthalmoplegia interna.

I think Dr. Gordon's statement as to the frequent mention of the Argyll Robertson phenomenon in the literature is correct. Adler, however, in reviewing this subject, greatly diminishes the number of these cases by stating that many transitory Argyll Robertson signs have been included in this list; he asks the question "Are we entitled to speak of transitory Argyll Robertson phenomena?" Perhaps such a term is allowable but I believe that, before cases are added to the literature as being true Argyll Robertson phenomena, even if transitory in nature, we should always be sure that we are not dealing with an ophthalmoplegia interna.

## FULMINATING ENCEPHALOMYELITIS\*

HENRY EDMUND MELENEY, M.D.

PEKING, CHINA

The clinical differentiation between typical cases of acute anterior poliomyelitis and typical cases of epidemic encephalitis ordinarily offers no problem. Even though there are no clear-cut differences between the microscopic lesions of the two diseases, nevertheless the distribution of the lesions, and the symptoms produced by such lesions, leave no doubt as to the correct diagnosis in any given case. When fulminating cases of either disease occur, however, an accurate diagnosis may be more difficult. In epidemic encephalitis fulminating cases are of rare occurrence, although the fact that they occur is mentioned by many writers on the subject.<sup>1, 2</sup> Such cases may show only general symptoms of an overwhelming infection, or there may be localizing symptoms which make the diagnosis fairly certain, as in the case reported by Leiner.<sup>3</sup> Fulminating cases of acute anterior poliomyelitis are also seen, but are common only during epidemics of that disease. These cases are mainly cerebral in type and may show, clinically, little or no evidence of involvement of the spinal cord. Microscopically, also, the brain is extensively involved while the spinal cord may show relatively few lesions. In such cases the simultaneous occurrence of typical spinal cases of poliomyelitis is the chief aid in establishing the diagnosis.

The epidemic of acute encephalomyelitis which occurred in Australia in 1917 and 1918, designated by Cleland and his co-workers<sup>4</sup> as the "Australian X-Disease," consisted almost entirely of cases of this cerebral type. The disease seemed to be a distinct clinical entity, because its geographical distribution was quite different from that of the typical cases of both epidemic encephalitis and anterior poliomyelitis which occurred at the same time. From an experimental point of view it also differed from poliomyelitis, since the investigators of the

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\* The study of these cases was made in conjunction with Professor Andrew H. Woods, head of the Department of Neurology, Peking Union Medical College.

1. Barker, L. F.; Cross, E. S., and Irwin, S. V.: *Am. J. Med. Sci.* **159**: 337 (March) 1920.

2. Gottstein, W.: *Ergeb. d. Hyg., Bakt., Immun., u. exper. Ther.*, **5**:394, 1922.

3. Leiner, J. H.: *New York M. J.* **112**:178 (Aug. 7) 1920.

4. Cleland, J. B., and Campbell, A. W.: *Brit. M. J.* **1**:663 (May 31) 1919  
This work is reported in detail in the Report of the Director-General of Public Health, New South Wales, 1917, Part V, Eighth Report of the Microbiological Laboratory for the year 1917, p. 144.

disease were able to transmit it to animals which have never been experimentally infected by the virus of typical anterior poliomyelitis. Flexner,<sup>5</sup> however, has preferred to consider the disease an aberrant form of anterior poliomyelitis rather than to accept it as a new disease on the evidence that has so far been offered. His view is strengthened by the fact that no subsequent epidemic of a similar nature has occurred either in Australasia or elsewhere.

The two cases here presented conform closely in all respects to the fulminating cases in the Australian epidemic, and to the cerebral form of acute anterior poliomyelitis, as well as to fulminating cases of epidemic encephalitis. They occurred at the same time, terminating within thirty-two hours of each other, and therefore gave rise to the suspicion that they might be the beginning of an epidemic similar to the one in Australia. No such epidemic actually occurred, and, since the cases were not known to have been in contact with each other, their close proximity in point of time was probably a mere coincidence. They are reported as interesting problems in diagnosis.

#### REPORT OF CASES

CASE 1.—*Clinical History*.—J. K., an Englishman aged 20, a soldier in the British Legation Guard, Peking, who had been in the Orient one year, three months before death had jaundice that lasted nine days. Two months before death he had had venereal sores which were cured by silver nitrate. His blood had showed a negative Wassermann reaction five weeks after these sores appeared. Aug. 29, 1921, eight days before death, while in camp at the seashore about one hundred miles from Peking, he was thrown from a donkey and landed on his head and shoulders. The next day he was able to go on all parades, and played football in the afternoon. September 3, three days before death, he played rugby football in the morning and later bathed in the sea and went to sleep in the sunshine. He felt "queer" that evening when his company started to return to Peking. He traveled all night on a train. On arrival in Peking, September 4, he was admitted to the Legation Guard infirmary; the temperature then was 105 F., the pulse was 130 and respirations were 31. He complained of frontal headache and of dorsal and lumbar pains. He was mentally dull. There was a macular skin eruption over the entire body including the limbs. The patellar reflexes were slightly exaggerated. September 5, the mental torpidity increased; the temperature was 105 F., and the pulse was 124 and weaker. He passed urine involuntarily. Spinal puncture on this day showed clear fluid, the centrifugized sediment of which contained only a few polymorphonuclear and mononuclear leukocytes. Râles appeared in both lungs. The Kernig and Babinski signs were negative. There was no ankle clonus. In the evening muscular rigidity developed, and a slight strabismus. The head was not retracted. Urine and feces were passed involuntarily. September 6, his temperature remained high, the muscular rigidity became less and the skin eruption disappeared. Dulness on percussion and râles developed over both lungs posteriorly. Fifteen cubic centimeters of spinal fluid was withdrawn but

5. Flexner, Simon: In the Symposium on Epidemic Encephalitis, New York Academy of Medicine, May 20, 1920, Med. Rec. 98:705 (Oct. 23) 1920.

showed no change from the former findings. The urine was normal. Coma became progressively deeper and the patient died at 7:30 p. m., just three days after the onset of acute symptoms.

*Necropsy Findings.*—Necropsy was performed thirteen and one-half hours after death. The viscera were normal except for general congestion, fibrous adhesions of the upper lobe of the right lung to the chest wall, patches of lobular pneumonia in the right lower lobe and in both lobes of the left lung, a few patches of hemorrhage in both lungs and in the mucosa of the stomach, slight distention of the urinary bladder and slight atheroma of the aorta. The meningeal, cerebral and spinal blood vessels were all intensely congested. The meninges were normal except for slight thickening at the base of the brain, where the cerebrospinal fluid seemed a little cloudy. There was no evidence of acute meningitis. On gross section the brain showed marked congestion but no gross hemorrhages or other lesions. In the spinal cord the gray matter of the cervical and dorsal regions appeared irregular and there were spots suggestive of hemorrhage.

Bacteriologic examination was limited to culture of the heart's blood, owing to lack of facilities at the place where the necropsy was performed. This culture yielded a gram-negative bacillus, morphologically and culturally similar to the glanders bacillus. It failed, however, to produce the typical glanders lesions in a male guinea-pig, and was therefore considered probably a contaminant.

*Histologic Findings.*—Microscopic sections of the viscera corroborated the gross findings and, in addition, showed pus in the small bronchi, areas of hemorrhage in the spleen, and infiltration of the portal spaces in the liver by many mononuclear cells.

The microscopic sections of the brain and spinal cord showed very extensive and intense lesions. These lesions consisted of: (1) Focal accumulations of wandering cells associated with rarefaction of the parenchyma; (2) phagocytosis of nerve cells, particularly of the cells of cranial and spinal nerve nuclei; (3) degeneration of nerve cells; (4) perivascular accumulations of wandering cells, associated often with a broad open space about the affected vessels; (5) diffuse infiltration of the parenchyma of some regions of the brain by wandering cells.

Of these lesions the focal cell accumulations were the most prominent and most extensive. They occurred in practically every section examined, including frontal, parietal and occipital cortex, thalamus, corpus striatum, midbrain, pons, cerebellum, medulla oblongata, and cervical, thoracic and lumbar spinal cord (Figs. 1-4). They were largest and most numerous in the midbrain, and smallest in the cortex. They occurred usually in the gray matter, but sometimes also in the white matter beneath the cortex, and in tracts in the midbrain. In the cervical region of the spinal cord they occurred in the posterior horns of gray matter as well as in the anterior horns. The cells found in these accumulations were mostly of the large mononuclear phagocyte type, with round, oval or irregular nucleus and pale-staining cytoplasm. Some may have been the so-called "ameboid glia cells." There were a few lymphocytes and polymorphonuclear leukocytes.

These focal accumulations of wandering cells were associated with rarefaction of the tissue in which they occurred, so that often only a loose network of fibers remained. In no case, however, was there complete dissolution of the tissue nor destruction of the phagocytic cells themselves, such as would occur in an abscess. The nerve cells seemed to be the only cellular elements to suffer.

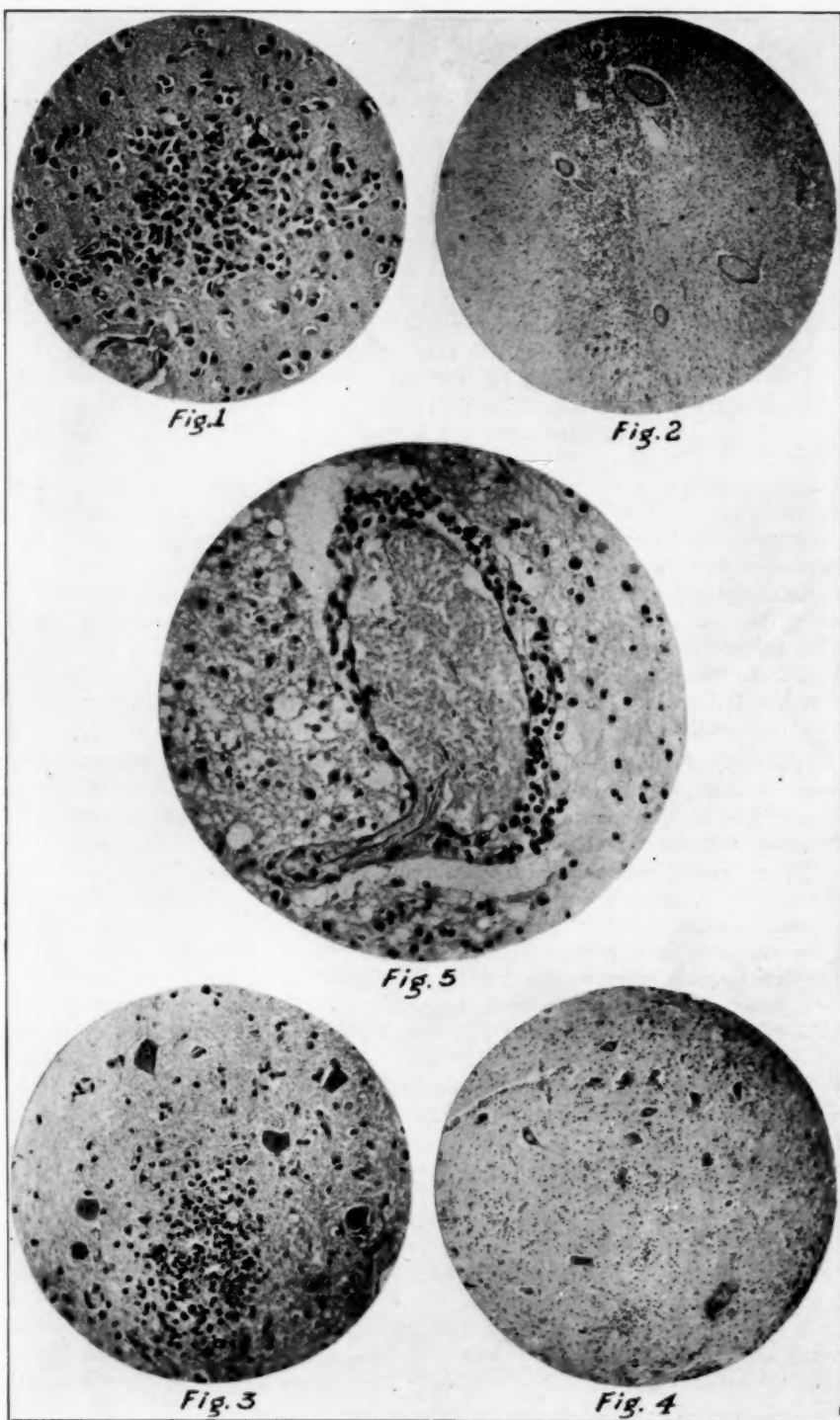


Fig. 1. Case 1.—Focal cell infiltration in gray matter of occipital cortex.  $\times 157$ . Fig. 2. Case 1. Focal cell infiltration, with rarefaction of the parenchyma, in the midbrain, region of third cranial nerve nucleus.  $\times 20$ . Fig. 3. Case 1. Focal cell infiltration in dentate nucleus of cerebellum.  $\times 120$ . Fig. 4. Case 1. Focal cell infiltration and neuronophagia, anterior horn of cervical spinal cord.  $\times 40$ . Fig. 5, Case 1. Perivascular infiltration of lymphocytes in the midbrain.  $\times 230$ .

The cells which had accumulated about blood vessels, as well as those diffusely infiltrating the parenchyma, were mostly lymphocytes (Fig. 5). Neither of these were striking conditions, the perivascular infiltrations being notably less than is usually seen in either acute poliomyelitis or in epidemic encephalitis.

Phagocytosis of nerve cells was, in places, very pronounced, particularly in the midbrain, the medulla oblongata, and the cervical spinal cord. The phagocytic cells were almost all of the large mononuclear variety (Figs. 6 and 7).

Degeneration of nerve cells included all stages of the process, from slight swelling and loss of Nissl substance to complete loss of nucleus and fragmentation of the cell body. In most places the degeneration was relatively slight, but in the midbrain, pons and medulla it was marked, and in the cervical cord it was extreme.

The pia of both brain and spinal cord was but slightly involved in the disease process. There was a little swelling of the fibrous network in places, and accumulation of a few lymphocytes about some of the blood vessels.

No perivascular hemorrhages were found in any of the sections studied.

*CASE 2.—Clinical History.*—Mrs. G. F., American, aged 56, had arrived in Peking from the United States about a week before her death. Her past history was negative except that she had suffered, at times, from violent headache and once had had a severe streptococcus throat infection. She had been vaccinated against small pox unsuccessfully in Japan two weeks before her arrival in Peking. Six days before death she had a sore throat but did not see a physician. The following day she complained of headache and of "fulness" in the right ear. She was seen by Dr. Willner of this department, who found that her throat was inflamed, and that there were a few coarse râles in the lower lobe of the right lung. Temperature was normal. Two days later (three days before death) she was seen again by Dr. Willner. Although she said that she felt well, her urine showed a small amount of albumen and a few red and white blood cells, but no casts. That night she did not sleep well; the following morning she had a temperature of 104 F., and vomited twice. She was then admitted to the Peking Union Medical College Hospital (Sept. 6, 1921).

On admission (two days before death) she was drowsy and mentally "cloudy." There was an erythematous blush on her chest, and several maculopapules were present on the abdomen and arms. Conjunctivae and throat were slightly injected. There were a few crackling râles in the base of the left lung. Reflexes were normal. Temperature was 39.6 C., pulse 90, respirations 26. Urine showed a large amount of albumin, a slight reaction for acetone, many hyaline, epithelial and granular casts, and a few red and white blood cells. Hemoglobin was 85 per cent.; white blood cells were 12,700 per c.mm., with polymorphonuclear leukocytes 71 per cent.; blood pressure, systolic 190, diastolic not recorded.

September 7, patient went into deep coma and became cyanotic. There was slight edema of the dependent parts of the body. Diffuse coarse râles appeared in both lungs. A systolic murmur appeared over the apex and base of the heart. Urine was not passed voluntarily but 800 c.c. was removed by catheter. Blood urea was 26 mg. per 100 c.c. Throat culture showed a non-hemolytic streptococcus. Blood culture was contaminated. Temperature 40.6 C., pulse 120, respirations 36. The patient died September 8, at 3:15 a. m. The clinical diagnosis was: Acute exacerbation of a chronic nephritis. No suspicion of acute encephalitis was entertained.

*Necropsy Findings.*—Necropsy was performed twelve hours after death. The body was that of a very obese woman. The conjunctivae were quite edematous and there was a suggestion of slight general edema. The viscera were normal except for two small hemorrhages in the endocardium of the right ventricle, slight thickening of the cusps of the aortic valve, atheroma of the coronary arteries and aorta, atelectasis of the lower lobes of both lungs, acute bronchitis, a small area of consolidation in the right lower lobe, slight narrowing of the cortex of the kidneys with a granular appearance of the subcapsular surface, slight thickening of the renal arteries, and several hemorrhages in the left tonsil. There was marked congestion of all the viscera. At this point in the necropsy the probable cause of death was thought to be either uremia or septicemia. Therefore the head was injected with ten per cent. formaldehyd solution before being opened, thus precluding the possibility of making cultures or animal inoculations from the brain. The meninges were not thickened. The brain, though injected with formaldehyd, still appeared markedly congested, but was not edematous. On gross section no lesions of any kind were revealed. There was hemorrhage into the bone cells above the right tympanic cavity. The spinal cord was grossly normal.

Cultures made at necropsy from the heart blood and from the spleen showed no growth.

*Histologic Findings.*—Microscopic sections of the viscera corroborated the gross findings and, in addition, showed occasional hyalinization of heart muscle fibers, hemorrhage into some of the alveoli of the lungs and edema in others, hemorrhages into the spleen pulp, chronic passive congestion of the liver with fat infiltration, degeneration of the cells of the renal convoluted tubules, moderate thickening of the small renal arteries, and acute inflammation of the left tonsil and of a nearby lymph node.

The microscopic sections of the brain and spinal cord showed, in general, the same types of lesion as those found in Case 1. All the lesions were, however, less intense and less extensive. The cerebral cortex showed no histologic lesions except an indefinite degeneration of the large pyramidal cells. The most marked lesions were in the midbrain, pons, medulla and cervical spinal cord, with less involvement of the thalamus, internal capsule, cerebellum and thoracic spinal cord. Focal accumulations of wandering cells, mostly of the large mononuclear type, were again the most prominent lesions (Fig. 8). Neuronophagia was less extensive, but, in places was intense (Fig. 9).

Perivascular accumulations of lymphocytes, however, were more prominent than in Case 1 (Fig. 10), though even here they did not reach the degree often found in cases of either epidemic encephalitis or acute poliomyelitis. As in Case 1, no hemorrhages were found either about the blood vessels or elsewhere in the parenchyma. The pia showed about the same degree of involvement as it did in Case 1.

#### COMMENT

From a clinical standpoint neither of these cases was accurately diagnosed. In Case 1, acute poliomyelitis, epidemic encephalitis and insolation were all considered, but the case was typical of none of them. In Case 2, the age, high blood pressure and urinary findings drew attention particularly to the kidneys. Coma was the only symptom referable to the central nervous system, and that symptom could have accompanied any overwhelming infection or the late stages of

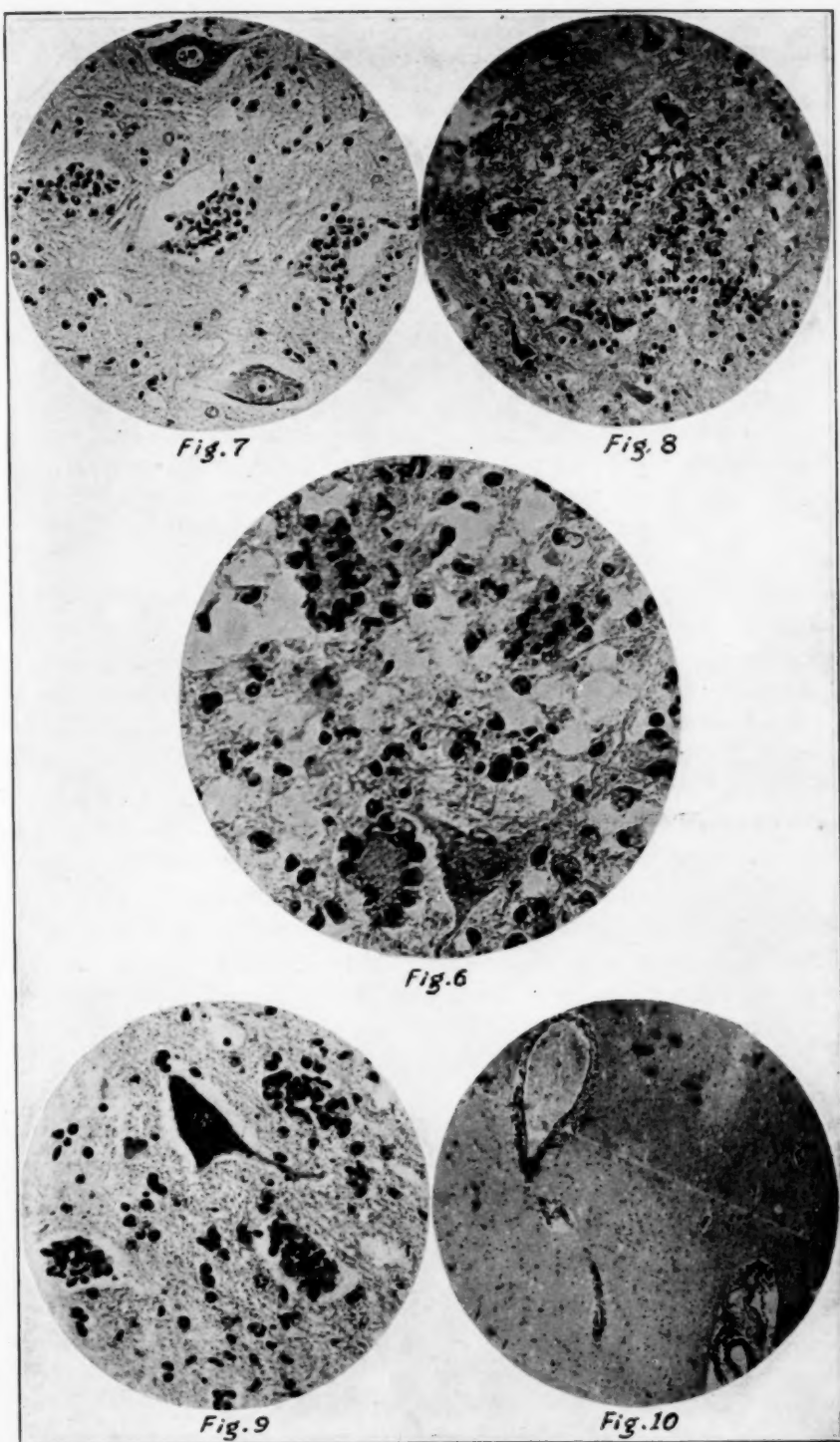


Fig. 6. Case 1. Neuronophagia in the midbrain.  $\times 310$ . Fig. 7. Case 1. Neuronophagia of anterior horn cells of cervical spinal cord.  $\times 180$ . Fig. 8. Case 2. Focal cell infiltration in the pons.  $\times 157$ . Fig. 9. Case 2. Neuronophagia of anterior horn cells of cervical spinal cord.  $\times 210$ . Fig. 10. Case 2. Perivascular infiltration of lymphocytes in the pons.  $\times 40$ .

uremia. Both cases terminated so rapidly that the element of time, which is often so helpful in clearing up obscure cases, was of no aid in making a diagnosis. In Case 1 the symptoms were more definitely cerebral and the microscopic findings were more intense and extensive than in Case 2. Had the patient in Case 2 lived another day, the symptoms and lesions might have corresponded more accurately than they did with those in Case 1.

Microscopically both cases show, in general, lesions which are typical of both epidemic encephalitis and anterior poliomyelitis. Perivascular lymphocytic infiltrations, focal and diffuse accumulations of mononuclear wandering cells, nerve cell degeneration and neurophagia are all found in typical cases of both these diseases. The one lesion which is commonly found in these diseases and which was absent in the cases here reported is perivascular hemorrhage, but it is not a constant finding, and might have appeared in these cases if the patients had lived longer. The focal cell accumulations, on the other hand, are usually not so prominent in typical cases of encephalitis or poliomyelitis as they were in these two cases. In places they almost gave the appearance of microscopic abscesses, although the preservation of neuroglia and infiltrating cells, together with the destruction of nerve cells, differentiate the lesions from those usually produced by pyogenic organisms.

Through the kindness of Prof. J. B. Cleland, the opportunity has been presented to compare the microscopic sections from these cases with sections from cases of the "Australian X-Disease." The lesions in our cases corresponded quite accurately, except for their greater intensity, to those in a human case of the Australian disease, but did not conform so closely to the lesions in the tissues from Cleland's experimental animals, sheep and monkeys, where the lesions were much less marked, and where perivascular infiltration of lymphocytes was the most prominent abnormality.

The possibility cannot definitely be excluded that one of the ordinary pyogenic bacteria was the responsible agent in one or both of our cases. Unfortunately it was impossible to take cultures from the brain and cord of Case 1; while in Case 2, the supposed nephritic etiology caused the omission of this procedure at necropsy. No bacteria were found, however, in any of the sections studied.

#### SUMMARY

1. Two cases of fulminating encephalomyelitis are reported from Peking, both of which resembled, clinically and microscopically, those occurring in the epidemic of so-called "Australian X-Disease" of 1917 and 1918.

2. The patients each survived only three days after the onset of acute symptoms, and died on consecutive days. There was no known contact between them. No epidemic of similar cases has occurred in this vicinity, as far as we know.

3. Microscopically the lesions consisted of focal accumulations of mononuclear and a few polymorphonuclear leukocytes, neuronophagia, perivascular and diffuse infiltration with lymphocytes, and nerve cell degeneration. The whole brain stem and spinal cord were involved in both cases, and in one case the cerebral cortex was involved as well.

4. An accurate etiologic diagnosis of the cases is impossible. They may have been fulminating cases either of acute anterior poliomyelitis or possibly of epidemic encephalitis. There is also a possibility that the etiologic agent may have been one of the more common pathogenic bacteria.

## SPINAL SUBARACHNOID BLOCK

ITS SIGNIFICANCE AS A DIAGNOSTIC SIGN.  
ANALYSIS OF FIFTY-THREE CASES \*

JAMES B. AYER, M.D.

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In two earlier papers<sup>1</sup> the writer has described a method by which it is possible to demonstrate spinal subarachnoid block in cases of compression of the spinal cord and other conditions which obstruct the free interchange of fluid within the spinal subarachnoid space. The object of the present short communication is to review briefly the results of this method after nearly four years of clinical use, in order to form some estimate as to its value.

The method employed has remained unchanged with certain modifications. It depends on (1) a careful manometric study of the fluid, by means of double and at times of triple puncture of the subarachnoid space, and (2) comparison of the fluids obtained at two or more loci.

For the hydrodynamic studies the points of puncture are designed to be above and below the obstruction, and therefore needles are not necessarily inserted at the same loci in all cases; for supposed cord tumor, cistern-lumbar puncture is the rule; for possible cauda tumor, double lumbar puncture may be preferred. Dynamic studies in low cauda tumor may be carried out also by means of combined puncture of the lumbar subarachnoid space and the sacral epidural space.

Concerning the second criterion on which the diagnosis of block is based, the examination of the fluid from two or more loci, experience has emphasized the significance of the protein content of the fluid as by far the most important. Normally, it has been found that the protein content of the fluid is almost the same throughout the spinal subarachnoid space. In obstruction, the protein has always been found increased below. Hence much reliance is placed on comparative quantitative estimations of protein in fluids obtained from different loci.

While fluids presenting xanthochromia, and more rarely the complete Froin syndrome, are present in a number of these cases, the majority of fluids from below obstructions have been found to be colorless. It is therefore certain that this method is more sensitive than is the examination of the lumbar fluid alone. The accuracy and significance of the information obtained will become apparent on study of the cases.

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\* Read before the Forty-Ninth Annual Meeting of the American Neurological Society, Boston, June 1, 1923.

1. Arch. Neurol. & Psychiat. 4:529 (Nov.) 1920, and 7:38 (Jan.) 1922.

COMPLETE AND INCOMPLETE BLOCK

In fifty-three cases it has been possible to demonstrate an interruption in the free interchange of fluid in the spinal subarachnoid space.

In forty-one cases the block appeared complete, i. e., on lowering fluid pressure distal to the obstruction by simple withdrawal, the pressure remained low even though that above the obstruction was three

TABLE 1.—Location of Block in Fifty-Three Cases

	Extradural	Meningeal	Intramedullary	Doubtful
Tumors—27 cases:				
Verified by operation.....	10	6	8	..
Verified by necropsy.....	..	1	..	..
Indicated by Roentgen ray.....	2	..	..	..
Potts' Disease—8 cases:				
Verified by operation.....	2	..	..	..
Clinical and Roentgen-ray diag..	6	..	..	..
Vertebral Dislocation—7 cases:				
Verified by operation.....	5	..	..	..
Verified by necropsy.....	1	..	..	..
Indicated by Roentgen ray.....	1	..	..	..
Subacute and Chronic Meningitis—7 cases:				
Verified by operation.....	..	2	..	..
Clinical diagnosis .....	..	5	..	..
Undetermined .....	..	..	..	4
Total.....	27	14	8	4

TABLE 2.—Spinal Subarachnoid Block: Complete in Forty-One Cases; Incomplete in Twelve Cases

	Complete Block	Incomplete Block	Total
Tumor: Extradural .....	7	5	12
Meningeal .....	5	2	7
Intramedullary .....	6	2	8
Potts' disease .....	8	0	8
Vertebral dislocation .....	6	1	7
Subacute and chronic meningitis.....	5	2	7
Undetermined .....	4	..	..
Total .....	41	12	53

TABLE 3.—Level at Which Block Occurred

	Cervical	Thoracic	Lumbar	Cauda	Diffuse	Doubtful
Tumor.....	8	14	3	2	..	..
Potts' disease.....	1	7	..	..	..	..
Vertebral dislocation.....	1	3	3	..	..	..
Meningitis.....	..	..	..	..	7	..
Undetermined.....	..	..	..	..	..	4
Total.....	10	24	6	2	7	4

or four times as great; conversely if pressure above was artificially raised by compression of the jugular veins, no compensatory rise was registered in the lower manometer.

In twelve cases the block appeared incomplete. In these cases the fluid pressure, changed in any manner either above or below the obstruction, tended to become equalized. In this connection it is probable that the changes noted after simple withdrawal of fluid

indicate more accurately the degree of block, but the results obtained by jugular compression are certainly more convincing.

Unfortunately, space does not allow a chart of all findings in these cases. A work-sheet (Fig. 1) will, however, give an idea of the

Consultant  
NAME Garfield Age 31 No. M.G.H. 248252 Date Mch. 3, 1922

Diagnosis (before c.s.f. exam.): ? Cervical rib. ? Tumor of cord.

Pain in right neck, forearm and hand for 2 years. No symptoms referable to lower extremities.

Brief clinical record: Exam.: Atrophy and weakness and sensory loss of right arm and shoulder. No weakness or sensory loss below. Reflexes lively; patellar clonus on right; plantar reflexes normal.

Block expected?

	Dr. Ayer Cisterna (4.3 cm.)	Dr. Townsend Lumb. (      space)	Dr. Other locus
Initial pressure	140 mm	140 mm	
Ampl. pulse osc.	2 mm	0.5 mm	
" resp. "	10 mm	3 mm	
Cough			
Strut			
Rise on jug. compression			
Fall " " release			
Rise on abdominal compression			
Press. before withdrawal	130 mm	130 mm	
" after taking 5 c.c. L	130 mm	70 mm	
" " " 5 c.c. C	75 mm	70 mm	
Rise on J.C. for 30 sec.	Prompt to over 240 mm	No change for 10 sec.; then slow rise to 150 mm	
" " " 1 min.	Prompt fall	Remains at 130 mm	
Release of jug. compress			
Character of fluid:	Clear, colorless; no clot.	Clear, colorless; no clot.	
Cells:	1	2	
Protein: alcohol ppt.	normal	mod'y increased	
ammon. sulphate	0	positive	
sulphosalicylic, mgrs.	20 mg per 100 cc	82 mg per 100 cc	
Wassermann	negative	negative	
Goldsol			

Opinion: Incomplete block.

Note: On Mch 11, 1922 laminectomy, C 2-6 vertebrae. Extradural fibroma measuring 4x5x5 cm removed.

JAMES R. AYER, M.D.

Fig. 1.—Shows method of tabulating data used in the study of cases in which spinal subarachnoid block is suspected.

method of study, and a few tables will summarize the most significant results (Tables 1, 2, 3 and Fig. 1). In Table 2 it will be seen that, of the fifty-three cases presenting block, we are certain of our diagnosis from operation in thirty-three, from necropsy in two, and are relatively

certain from roentgen ray and clinical studies in fourteen; in four cases only do we feel doubt as to the cause of obstruction. Analysis of these tables may be briefly summarized as follows: block, complete or partial was caused by tumors, by vertebral disease and dislocation, and by meningitis; block was produced by extradural, meningeal and intramedullary agents, and all levels of the spinal subarachnoid space are here represented.

From a consideration of these tables it appears that the demonstration of block by this method is a reliable guide as indicating obstruction

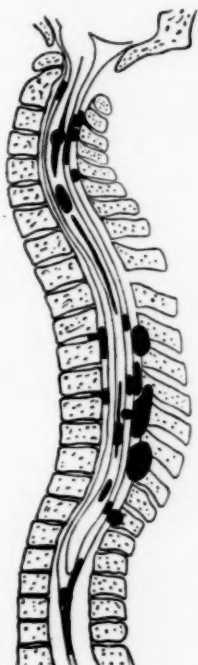


Fig. 2.—Diagram to show the level and location of tumors with reference to the spinal cord in which block was demonstrated.

to the free flow of fluid within the spinal subarachnoid space. Furthermore, the reliability of incomplete block appears to be as great as complete block, and its significance greater, in that it may be found in lesser degrees of compression of the cord than complete block. Unfortunately there is little in the study of these cases which indicates the level at which block exists, except when differential lumbar punctures have been employed; nor is any indication given as to the location of the lesion with reference to the cord and its membranes, or as to the type of lesion, except in meningitis in which triple punctures have been employed.

## SIGNIFICANCE OF PATENT SPINAL SUBARACHNOID SPACE

While the reliability of positive findings seems assured, can we attach any significance to negative findings? Especially important, is absence of block compatible with compression of the spinal cord?

In 104 cases it has been possible to demonstrate a freely permeable subarachnoid space. Not all of these patients were cord compression suspects, a number being under treatment for cerebral syphilis by the cisternal route. Table 4 represents diagnoses made after sufficient period of observation to render them accurate in most instances. Three

TABLE 4.—One Hundred and Four Cases in Which Block Was Not Demonstrated

	No. of Cases
Laminectomy for supposed tumor of cord; no tumor found.....	6
Laminectomy for tumor of cauda equina; tumor found (below low puncture)....	2
Laminectomy for tumor of cauda equina; tumor not found.....	1
Multiple sclerosis.....	15
Meningitis: acute.....	8
tuberculous.....	2
Neurosyphilis: acute meningitis.....	1
meningovascular (no obstruction found at necropsy in one case)....	15
general paralysis.....	3
tabes.....	2
optic atrophy.....	3
Combined system disease.....	3
Degenerative myelitis.....	3
Hematomyelia.....	1
Poliomyelitis: acute.....	1
chronic.....	1
Tumor of vertebra; no spinal cord symptoms.....	1
Trauma of spine; no spinal cord symptoms.....	1
Acute myelitis.....	1
Cerebral arteriosclerosis.....	1
Potts' disease; no cord symptoms.....	1
Hypertrophic arthritis of spine.....	2
Paralysis agitans.....	1
Progressive muscular atrophy.....	1
Encephalitis lethargica (one case confirmed by necropsy).....	2
Syringomyelia.....	1
No diagnosis permissible.....	22
Epidural cyst (found at operation).....	1
Intradural metastatic adenocarcinoma (found at operation).....	1
Extradural neurofibroma (found at necropsy).....	1
Total.....	104

groups chiefly concern us. First, six cases in which laminectomy was performed for supposed tumor of the spinal cord in spite of the fact that no block could be demonstrated. No tumor or other obstruction was found in any of the six. Second, attention is called to the large number of patients who presented the symptomatology of a transverse cord lesion and who subsequently developed unmistakable signs of multiple sclerosis, fifteen in all. Third, a group of three patients who presented pathologic lesions which fall into the group of "cord compression," but in whom block was not demonstrated.

It will be well to speak of these three cases individually. The first patient presented the picture of a complete transverse lesion of the spinal cord, with no noticeable change in symptomatology in ten years. At operation a multilocular cyst was found, about the circumference of

the forefinger, lying dorsally in the epidural space, and extending beyond the operative field both above and below. The origin of the cyst is problematic. The patient in whom adenocarcinoma was found likewise presented unmistakable transverse myelitic symptoms. In her case the spinal cord, instead of appearing swollen, was actually constricted a little at the point at which the tumor was found. The findings in the third patient were most instructive. More than a year previously he had had a sciatic neuroma removed. Because of severe root pain a spinal neuroma was suspected, although no signs of cord compression were present. While no block was demonstrable the fluid was definitely pathologic, showing 135 mg. of protein in the lumbar fluid and 30 mg. in the cisternal fluid. The pain was subsequently relieved by excision of a large intercostal neuroma; but at necropsy a short time later, an extradural neurofibroma, apparently silent as far as symptoms were concerned, was found.

From a study of these cases it is reasonable to conclude that a permeable subarachnoid space may be present in cord compression (three cases), but in that laminectomy was performed six times with negative findings, in the absence of demonstrable block, a certain value must be attributed to negative dynamic studies. One more point in this connection; while the three cases just mentioned showed no block, two of them did show marked increase in protein below the tumors as compared with the protein above. In that every case of the fifty-three in which block was demonstrated by dynamic tests also showed increase in protein below the block, it is reasonable to conclude that protein increase is a more delicate indication of cord compression or meningeal involvement than is demonstrable block.

#### CONCLUSIONS

It is certain, from the above records, that the demonstration of block is a reliable guide as to the permeability of the spinal subarachnoid space.

Conversely, while tumors have been found in patients in whom block was not shown, the evidence at hand suggests that this is rarely the case.

The writer and his associates have come to place more and more reliance on this physiologic sign in cases presenting transverse myelitic symptoms, and believe that it is the one most reliable sign we possess in distinguishing compressive from degenerative lesions of the spinal cord.

While careful dynamic studies may be carried out in most cases by the use of lumbar puncture alone, lesser degrees of block are unquestionably demonstrated with greater certainty by means of combined punctures.

Finally it is suggested that in every diagnostic lumbar puncture the aqueous manometer should be used, and dynamic studies carried out (especially the effect of jugular compression) as part of the routine examination of the spinal fluid. When block is partial or doubtful, combined punctures, as more delicate and certain in their interpretation than lumbar puncture alone, are indicated.

#### DISCUSSION

DR. LAURENCE B. SELLING, Portland: Any one who has used the method suggested by Dr. Ayer cannot fail to have been impressed by its value. I have had a small series of cases which have clearly demonstrated the value of the method. In five cases I have found complete block, with all the changes which he describes. Three were spinal cord tumors at various levels, verified by operation; one was a sarcoma of the fourth dorsal vertebra, invading the cord, and the fifth was a case of Pott's disease.

As Dr. Ayer states, the most valuable signs on the hydrodynamic side are the difference of pressure and the fact that the rise after compression of the jugulars is different at the two levels. It has been a matter of considerable interest that the rise on pressure is quite different from the rise on coughing. In the last case of spinal cord tumor which I had the opportunity of observing, jugular compression produced a marked rise of the cistern pressure and no rise at all in the spinal canal. On coughing, in the same case, there was an equivalent rise in the lumbar needle and in the cistern needle, showing that we cannot use cough as a differential sign at all. In that respect, it differs entirely from jugular compression.

One point is a little doubtful as yet, and that is the significance of the partial rises in pressure. Sometimes, on jugular compression, one gets a rapid rise at the cistern and a relatively slow rise in the lumbar needle. That has occurred in cases in which I had no reason to suspect even partial cord compression. I have had no way of checking it up as these cases did not come to necropsy. I do not know, however, that the slow rise in the lumbar needle can be used as a sign of partial block. This may occur in normal cases.





George M. Beard.

## DR. GEORGE M. BEARD

A SKETCH OF HIS LIFE AND CHARACTER, WITH SOME  
PERSONAL REMINISCENCES \*

CHARLES L. DANA, M.D.

NEW YORK

### INTRODUCTION

A great many interesting tales could be told and some have been told about breaking into New York. It is a great adventure. Dr. George M. Beard came to New York at a time when it was not as easy as it is now. He came a stranger although he was a Yale graduate, and met his college friends here. But he made no important hospital or college connections, except that he lectured at the University Medical College for two years on nervous diseases and electrotherapeutics, and held a clinic for two years at Demilt. He did not care for hospital and laboratory work.

He at once plunged with tremendous energy into four fields of activity, all of which were new, and all of which were held rather in suspicion by the conventional medical men of that day. These were electricity in therapeutics; functional neuroses; social psychology; and writing for the *North American Review*.

He studied almost for the first time seriously and enthusiastically the subjective side of man. He noted as symptoms such things as nervousness, diffidence, morbid fears, chilly feelings, blushing, and fatigue states. But he worked just as intensely and thoroughly over these phenomena as if he were sectioning a cord or studying objective things by the bedside. One summer he heard of the Maine "jumpers." He left his practice and traveled to Maine to study them. He became interested in hypnotism and for months he gave up half his days to examining the phenomena. He compelled the profession to accept it as a morbid reality. He investigated mind-reading and showed it was really a reading with the muscles—a form of kinetic interpretation.

He attacked fanaticisms in diet and drink and developed a "Cosmic Law of Intemperance." He wrote of longevity and work, of brain workers and their health, and of the temperamental nervousness of social groups. He very early described neurasthenia, which became a household commodity later. And for ten years he exploited electrotherapeutics. He founded and published for two years a journal of neurology and electrology.

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\*An address delivered at the Forty-Eighth Annual Meeting of the American Neurological Association, May, 1922, Washington, D. C.

He appealed for better methods of medical education and laid down, forty years ago, views of medical education that are being reached and realized today. He wrote a novel—what is more serious, he destroyed it. He worked with Edison for a time on what they thought was a new force. They could not demonstrate it, but it is said now that what they came near discovering was the Hertzian wireless waves.

He was ignored, criticised, attacked, and yet much beloved during the sixteen years of his professional life.

#### BIOGRAPHICAL SKETCH

Dr. Beard was born May 8, 1839, in Montville, Conn. His father was a clergyman and his grandfather a doctor. He taught school for two years, then entered Yale College in 1858, and graduated in 1862. He studied at Yale Medical School for a year, then, in 1863 and 1864, he was assistant surgeon in the Navy. He graduated from the College of Physicians and Surgeons in 1866, at the age of 27, writing as a graduation thesis his essay on longevity and work. He then started his career in New York.

He had met Dr. Rockwell as a medical student. They both belonged to Alpha Delta Phi, and they became friends. Beard, after a time, proposed that Dr. Rockwell and he should have offices together. Dr. Rockwell was not quite decided; but one day he looked out of the window, and there he saw Beard with a wagon-load of his office equipment, and the compact was sealed. He began his career strategically from an office in Brooklyn, but, in 1868, he had established himself in Manhattan.

He married early a charming and attractive-looking lady, and had a pretty little daughter whom I can just remember. In his early years, Beard had financial struggles, and at one time planned to give up practice and become a lecturer. He even hired a hall in Harlem and tried it, but he had only the faithful Rockwell and the janitor as auditors. He gave it up.

His life was one of continuous activity and production, but there were no dramatic incidents. He practiced in New York, traveling often to Europe. In January, 1883, he died suddenly of embolic pneumonia.

Beard loved literary work and was at it continuously. He was quick to acquire knowledge. Up to the time of his medical graduation he knew no German; but having, in 1868, received an offer to translate Tobold's "*Chronic Diseases of the Larynx*," he set to work and in six weeks was able to undertake the task, and he did it.

In the next year he wrote a work on Domestic Medicine, for which he received \$800. He immediately started for Europe and came back

in three months with only fifty cents. Beard had no idea as to money except that it was to be spent. However, he eventually paid all his debts and in the last ten years of his life was prosperous.

#### PERSONAL CHARACTERISTICS

In 1880, Dr. Beard was 41 years old and at the height of his activities. It was at this time I made his acquaintance. I took an interest in the things he was doing and he took a liking to me; so that in the summer of 1881 when he went to Europe, he installed me in his office; and he did the same the following year.

Dr. Beard was not a shut-in type—perhaps no character could be more open in method or habits. He did not even practice in private, but would invite physician friends to come into his office during his morning hours and see his patients, and hear him question them and see him treat them. I went in one morning. A healthy but nervous and embarrassed young man sat in the patient's chair with his eyes on the ground. He would not look at Beard or me. This was the first time I had seen or heard of "anthropophobia."

Dr. Beard was a little deaf, which handicapped him, but did not embarrass him. Nothing could or ever did disturb his serenity. He lived in a kind of enchanted atmosphere of synthesizing mental activity. He was a man of slender build and moderate height, with a grave and placid face. I never saw him angry and he became eloquent and earnest, but never excited. He had a continual undercurrent of humor and met all his critics and all assaults on his character with a joke or with philosophic calm. He always thought he was right in his scientific conclusions and social views. He argued for them, but never quarreled. He believed in electricity, but when one day his battery did not work, he continued his applications with the dead electrodes and discovered psychotherapeutics.

He never indulged in personal criticism or said an unkind word against any one. He was very social in a way; he loved to meet human beings—any one—and to talk and listen. He was a patient listener.

His practice was never large—that is, never enormous. He kept no books, except a history book. This was in the form of an old ledger. The name and history were entered in an execrable handwriting. Then he would make an agreement to treat the patient for a certain time, say three or six months, for the sum of \$100 or \$300. This sum was entered in the left-hand upper corner, and when the patient made a payment, the sum was credited under it. I never saw any bill-head or account-book.

Dr. Beard was intensely therapeutical and believed in drugs and instruments, and in anything that would help to cure. He had a long list of special and often ingenious and unusual formulas, which

were put up for him in certain drug stores. He never handed out his medicines.

He had all kinds of devices for his neurasthenics: He used deep injections into the urethra, cold sounds and local electricalization. These were the days of Dr. F. N. Otis and the urethra; its stricture and irritations were much in evidence.

While intensely interested in hypnotism, he did not use it therapeutically. He believed that trance states were producible by many different methods, that they occurred spontaneously and were mildly morbid phases of psychic activity. His explanation of trance states was rather simple. (All his applied psychology was rather simple.) Trance was a state of intense attention—the attentive functions of sensation and perception were greatly increased in power, the other functions of the brain correspondingly lowered. Psychology was a baby science then, and Beard's psychology was more a social than an individual phase of it.

Dr. Beard was a man of temperate habits, but not ascetic. He liked at least to lunch well and he used to lunch at Delmonico's, which at that time was at Twenty-Third Street. I was not lunching at Delmonico's in those days, but Beard would take me with him and discourse on the topic he was then most interested in. Once at Delmonico's while eating a coldcut, he discoursed on diet. His views on diet were unique and probably wrong. The earth he said fed on gases and got its growth from them; the plants fed on the earth, the animals fed on plants. Among the animals, the vertebrates or higher fed on lower types. The higher type thrived best on types nearest to it but below it. Theoretically, the best diet for men was those animals nearest to man. He claimed that monkeys were good to eat. And he was at some pains to show that cannibal tribes were the most intelligent among savages! Meat he said was the soul of the neurotic diet. As a matter of fact red roast beef is a stimulant to depressed neuropathics—if they can handle it—and if they are not at the involuntional age.

Beard had a great fondness for music and dancing. At the age of 40 he would slip off of an evening to a dancing class for the pleasure of the exercise. He admired vocal music and prima donnas: "The queens of song are always pure," he said, meaning that prostitution spoiled the voice.

He wrote an essay on the "Cosmic Law of Intemperance." This law, announced by Bowditch, is that intemperance is least at the equator and increases as one gets toward the pole. Beard's law is that drinking is the same the world over, but the bad effects of drinking are seen least at the equator and most as one goes north.

Beard believed that the most powerful ally of the temperance cause was the nervous sensitiveness of the brain-working classes, which compelled them to abstain from alcohol as well as tea and coffee. He did not believe in prohibition.

He believed in the education of women and lectured sometimes at certain women's colleges. But on one occasion after lecturing at Smith, he said with a humorous grin: "I had to say to the audience of young women, most of whom were planning to earn a livelihood—woman loses her charm as soon as she begins to work for her living."

Beard's attitude toward money was almost pathologic. He was for years in debt, but he seemed rather to enjoy it.

He wrote an autobiography covering many of his earlier professional years from which<sup>1</sup> I extract the following:

"I congratulate myself that few persons at my time of life have succeeded amid severe disappointments, in honestly acquiring so admirable a band of creditors. In that select circle are found names, of whom, if the world is worthy, certainly I am not.

"It has been said by those who regard themselves as wise that you must winter and summer with one before you can know him, but I will recommend a shorter and surer road to acquaintanceship—the getting in debt to a man or allowing him to get in debt to you. Such delicate relationships bring out the finer, subtler and least suspected qualities of human nature, such as would never reveal themselves by any other test whatever; indeed, no man can be said to know himself, until he has been either a debtor or creditor."

Beard was aphoristic in conversation and I always left him with the feeling that I had been enriched by some stimulating phrase or thought. But he made no attempt at smart writing. He himself had a smooth and finished style—without any flights. He believed in the old college advice, "If you wish to get a good style, study Addison and the Spectator."

#### SPECIAL SCIENTIFIC INTERESTS

Dr. Beard took up a theme and proceeded to work at it till he had exhausted it; studying, writing, lecturing. He never dropped his tasks finally, till they were worked out.

(1) He wrote on electrotherapeutics for ten years—then stopped absolutely. Perhaps there was nothing more for him to say. More likely, it was because this was his pot boiler. I don't think he cared for it as for his other pet interests. He believed in it as he did in bromids and cold sounds, but I never heard him discuss electrotherapy.

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1. Abstracted from A Sketch, by A. D. Rockwell, M.D., Medical Record 24:401 (Oct. 13) 1883.

(2) He was very fond of his "longevity and work" thesis, and kept at it for twelve years, writing his final paper in 1878. In it he aimed to show that the original work is done before 60. Linked with this was his view, that brain work was healthy, that it was defensive against old age, that brain workers lived fifteen years longer than the average, that the higher the type of mental work, the healthier it was. Philosophy being the highest type of brain work, philosophers were the longest lived—and he gives statistics. Philosophy in the sense that he used and followed, was the continuous pursuit of knowledge and truth, a calm Olympic attitude toward the actions of human beings, a sane view and restrained interpretation of life. It was the highest of callings and led to happiness as well as long life. Beard practiced what he preached. He was always in good spirits, and, I think, happy, but no philosophy can withstand septic infection. So there:

"Came the blind fury with the abhorred shears  
And slit the thin-spun life."

(3) A third subject was hypnotism, or as he preferred to call it, trance. He became interested in this in 1874 and 1875. In connection with it, he visited the Eddy brothers and studied the spiritualists and mind readers. In 1874, there appeared a clever man named Brown, a mind reader who captured the town and even won over the faculty of Yale, with whom Beard had a famous and formal controversy. Beard contended that mind reading was muscle reading. He wrote an article in the *Medical Record* to this effect. He investigated the famous clairvoyant, Bishop, who achieved a great reputation the following year. In 1881 he took up hypnotism again.

His studies on this then tabooed subject were most ingeniously carried out. He did not write a paper or start a therapeutic clinic. But he had cases demonstrating various phases of trance exhibited and studied every other afternoon in his office; he invited friends and critics to come in. And we all came—those who believed—and those who did not. Dr. Birdsall and Dr. Seguin, Dr. Putnam-Jacobi, prominent surgeons and practitioners. His office was a clinic for several months. He pried into all the odd phases of his subject. I went with him once to hear an entranced spiritualist and embryonic Patience Worth, who poured out rhymed verse on any topic suggested to her. I remember she gave me a stunning pastoral poem on "The Cow." I went with him also to see a professional hypnotizer hypnotize his wife, who then, with eyes closed and bandaged, read the cards we handed her. We all became convinced that there was such a thing as hypnosis, and several venturesome surgeons did minor operations under hypnotic sleep.

The profession has never taken the same antagonistic and skeptical attitude that it held before those days.

In connection with hypnotism and his psychologic theory of its mechanism, he worked on his explanation of the Salem witchcraft and his laws of human testimony. He enumerated the factors by regarding which the fallibility of testimony could be lessened and observation of phenomena be protected against error. Beard did not have time or opportunity then to section the medulla.

(4) Nervousness, nervous exhaustion and the symptomatology of what we now call psychoneuroses was his longest and deepest love. He wrote his first article in 1868, which was published in the *Boston Medical and Surgical Journal*, April 29, 1869. It was not noticed for five years in the United States, but was taken up by Dr. H. Campbell of London and by Erb and other German writers.

Beard came back to it in 1879<sup>2</sup> and published his "Neurasthenia" in 1881.

Along with this study of the minor mental disorders ran his interest in the specific nervousness of the American people. On this subject he wrote his latest—and probably his best—book, in 1882. It is still interesting reading.

The word nervous and the term nervousness do not seem to have been in the vocabulary of the American people until after the Civil War. Then it was independently described by Beard, in 1868, and later by Weir Mitchell in 1877 (*Medical News and Library*, December, 1877). The latter reports a number of cases representing a clinical picture new to him. It was functional in character and he called it "nervous debility."

Beard wrote about nervousness all his life and was specially interested in it as a racial condition, calling for recognition and attention from physicians and the public. He writes an introduction to his book on this subject which is almost passionate in tone, pleading for a study of the nervous make-up of the American people as a matter of importance to its future.

He seemed to feel that his subject at that time had not much appeal and that a study of racial traits and reactions was then thought hardly worthy of scientific attention. For he says:

"My interest in this subject is justified:

"It is the function, the life of science to ennoble the small, to give importance to what seems trivial."

Beard's plea for a consideration of the nervousness of Americans, has something in it of that quality which has pervaded the tractates of the National Committee for Mental Hygiene and the appeals of Dr. Stewart Paton in the last few years.

2. *Medical Record*, 15:184, 1879.

He wanted a study made of man's behavior as affected by neural conditions. "The anxiety habit," he asserts, "is the cause and sign of nervousness." "The American," he adds, "is continually asking, 'How shall I make a living?' 'Who will be the next President?' 'Where will I go when I die?'"

Toward the end of his life he became deeply interested in medical education. Somewhere in the archives of the *Medical Record*, is a posthumous essay on the subject. The editor held it back, I think, as being too advanced and academic. However, Dr. Beard held views which may be called those of advanced eclecticism. He believed in the thorough scholarship and laboratory and clinical training of the Germans, but he especially insisted on the early bedside method of the French. "We should get our knowledge first through the senses." "Medical education begins at the bedside and ends at the laboratory and systematic text-books."

Some such combination of the French and German methods is being developed today. He believed in sound and thorough training, but not in German erudition. He says: "There is very little in the world really worth knowing and ignorance may be power as well as a joy." "Not knowledge, but the power to acquire and use it, is the supreme need." "Much knowledge is only for temporary use."

Probably Dr. Beard's greatest merit was his contribution and his stimulus to the study of the subjective symptoms and social phases of disease. He was accused of making people introspective and hypochondriacal through this method. What would the fathers of those days say to the methods of introspection and self-analysis which have become approved today? From his earlier method has evolved the view we now hold, that one must know profoundly and intimately the whole personality, in order to deal with the patient's psychosis, or his conflicts and maladjustments.

To me Beard appealed most because he saw things from the standpoint of an observant, original and humorous, yet, on the whole, a sound and progressive philosopher. He not only observed acutely, but he saw some general law behind everything and he freely threw off his generalizations, which were touched with humor and were often meant to be suggestive, rather than final.

He had plenty of occasions to test his philosophical calm, but it never failed. He had the mood of the man who was

"Integer vitae scelerisque purus."

He met the turbulence of life and the reactions of his fellow-men with a humorous tolerance or philosophic calm. He had no readjustments or maladjustments to labor over. These were left to his environments—to his friends and his critics.

And when he found that death, the cruelest test of all, was calling him at the ripest period of his activities, with many problems to be attacked, with friendship secured, with professional and economic success achieved, he uttered no complaint.

Many friends called during his last illness, and when he knew he was dying, he said to me at this time, that he wished his friends would continue his work; and he wished he were "strong enough to dictate the feelings of a dying man. It would be a most interesting contribution to science and psychology." Four hours later he passed away.

## FOLIE A TROIS\*

F. J. GERTY, M.D. AND GEORGE W. HALL, M.D.

CHICAGO

On March 10, 1923, three patients, two brothers and a sister, all alleged to be insane, were brought to the Cook County Psychopathic Hospital. The three had been living in the same apartment with the wife of the elder brother, to whom he had been married about three years. Another brother acted as janitor for the building and lived in the basement. This brother and his wife stated that mental symptoms were noticed first in the elder brother about four months before and that the sister and younger brother first showed evidence of mental disturbance about two months later. All three had been very greatly depressed for one month. At times all of them had been agitated and the household had been sleepless most of the time. The elder brother spoke of suicide and the sister had twice placed a rope around her neck to hang herself. Shortly before admission to the hospital the sister made a desperate attack on her brother's wife whom she seemed to regard as responsible for all the trouble that had come to the family.

The family history revealed little of value. The mother and father had been dead for many years. It was said that the mother had been subject to attacks of depression, but nothing more definite was learned. The brother, aged 60, who supplied most of the information was rather seclusive and lived alone in the basement of his sister's building doing the janitor work. His meals were prepared in his sister's apartment but he ate, by his own preference, after the rest of the family had finished. Another brother lives in Nebraska and has not been heard from for some years. A fifth brother has been dead for seventeen years.

The two male patients, George (the elder) and Frank, had been in the retail grocery and market business for over thirty-five years. They had been reasonably successful until the last three years, during which business had fallen off. In October, 1922, they sold out for \$1,400 and the money was all used to pay debts. For some months the sister, Katherine, had loaned the brothers part of the money she received as rent for one apartment to help pay their store rent. However, the family was not by any means in dire straits. The flat building owned by the sister was clear of debt and had a value of \$12,000. The other apartment brought \$80 a month rent. The younger brother, Frank, obtained work as a butcher and earned \$40 a week.

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\* Reported from the Cook County Psychopathic Hospital.

About a week after George entered the hospital he told his wife that he had something to confess. He had married against the wishes of his brother and sister. Frank had always been the dominant character in the household and managed the accounts and business transactions at the store. The sister had owned a \$6,000 interest in the flat building, the remaining \$6,000 being held jointly by George and Frank. Frank was much angered by the fact that George's wife acquired a claim on the property through her marriage. He thought that the whole property should be transferred to his sister and after the marriage he proposed to George that this be done. As George had not informed his wife how the property was owned he agreed. To avoid the necessity of obtaining the wife's signature, Frank had the documents made out by a lawyer who dated them back before the marriage. In this manner, Katherine became sole owner of the property. As stated, George informed his wife of these facts after his admission to the hospital.

The three were never easy in their minds as to the legal aspect of the transaction. In addition to their dislike of their sister-in-law, Frank and Katherine were in continual fear that she might discover what they had done. During the two months preceding admission to the hospital, the idea began to grow on them that the house was lost and that because of what they had done they would be cast into prison. They believed that money in the bank was also involved and would be taken from them. The hatred grew so strong that the day before she was taken to the hospital Katherine violently attacked the unsuspecting sister-in-law.

#### REPORT OF CASES

CASE 1.—George C., aged 56, married, who had had a common school education, suffered from rheumatism when about 22, received a scalp wound which bled profusely at 32, and had typhoid fever at 36, was said to have been subject at intervals to mild periods of depression. He had never been talkative or hyperactive, but was slow and quiet, though pleasant and friendly with his customers. However, when about 40, he suffered from a period of deep depression during which he was considerably agitated. It was necessary to send him to a sanitarium where he remained for several months; he apparently made a complete recovery. His wife did not learn of this previous attack until some time after marriage.

About a month and a half after the sale of their business, George became much depressed. He sat and brooded, and reiterated, "Everything is lost." He said that he had broken the law, but would not tell his wife in what way. He was afraid the police were coming to arrest him. He said he could see a dreadful furnace into which "horrible objects" were being thrown and that he himself was to be thrown into it. He often looked up at a three story building in the neighborhood and talked about going up on it to jump off. He had some pimples on his scalp, which was nearly bald and scratched these until his whole scalp was excoriated. Then he began to tear at his clothes and his trousers were soon in ribbons. He did not sleep at night but paced

the floor. He would not change his clothes or bathe. On admission to the hospital he was passive but evidently depressed. His clothes were torn and untidy. His scalp was covered with scabs as the result of scratching and he repeatedly raised his hands toward his scalp, but stopped the motion when commanded to do so.

*Examination.*—Physical examination revealed no marked abnormalities. The patient was well nourished, even slightly obese. His scalp showed excoriations from scratching and many infected hair follicles. His blood pressure was 105 systolic and 70 diastolic. Neurologic examination was negative. The blood examination showed 80 per cent. hemoglobin, 4,100,000 red cells, and 7,500 white cells. The differential count was: polymorphonuclear leukocytes, 71 per cent.; mononuclear leukocytes, 25 per cent.; eosinophils, 3 per cent. Blood and spinal fluid Wassermann reactions were negative. The spinal fluid contained 2 lymphocytes per c.mm. The blood chemistry findings were within normal limits. The urine showed no sugar, albumin, or casts.

He was oriented for time, place, and person, and his memory for remote and recent events was good. Retention was only fair, because the patient did not attend to the test. General information, calculation, and judgment (as displayed by answers to stories from the Binet-Simon test) were all fairly good. He was in contact with his environment and knew he was being examined as to his mental condition.

In behavior, he was restless, picked at his finger nails and raised his arms to scratch his head. His general aspect was that of marked depression. He kept his gaze directed downward, did not smile and he was very slow in his responses. He said he did not wish to discuss his trouble as it was useless. He blamed himself and said he must be crazy. For over thirty years he had been in business and finally he had lost out; he had lost everything after working all his life. He had also "committed a great crime and sin" (At the first examination he told nothing further about this; later, as already stated, he told his wife about deeding the house); there was no hope for him; it was useless for the doctor to bother with him.

He admitted that he had felt like this once before, in 1908, and had been in Kenilworth Sanitarium for three months. He had been much afraid at that time. No evidence of hallucinations or paranoid trends were elicited.

CASE 2.—Frank C., aged 52, single, who had had a common school education and two years in an academy, was much worried, paced the floor, groaned, and muttered, "lost." He also said that the man for whom he worked had obtained possession of the house and that there was no use in working. Frank had been mentally alert, but always a little peculiar and of the two brothers had always been the dominator.

An accidental and interesting side light on Frank's character was obtained from Dr. I. B. Diamond. Frank, a Catholic Irishman, had been keeping company for four or five years with a Jewess. This woman consulted Dr. Diamond. She was aged about 34 and her relationship with Frank was apparently platonic. She was obsessed with the idea that her dead mother was in every man-hole or catch-basin that she saw. She could not pass these places until she had visually substituted someone else for her mother. She could not wash her feet or take a bath because the idea entered her mind that the mother was in the water. She made artificial flowers and it seemed to her that she twisted her mother into the flowers and it was necessary for her to take the flower apart in order to gain relief. The reason underlying her mental state was that

she had fallen away from the religious teaching of her mother and this caused her to feel that her acts—even to keeping company with a Christian—had cast her mother down into undesirable sewer holes, bath water, etc. Frank's faithfulness to her was remarkable. During two years, in which she gave him up to assuage her conscience, he haunted the neighborhood of her house constantly. When she allowed him to return, he came to see her two or three times a week. He bathed her feet for her because of her repulsion for this act, and washed out her stockings for her.

*Examination.*—The physical examination was essentially negative. The systolic blood pressure was 120 and the diastolic 80. There were no pathologic neurologic findings. Blood and spinal fluid Wassermann reactions were negative. The urine contained no sugar, albumin or casts. Blood chemistry findings were within normal limits. The white blood cells were 5,200 per c.mm. and the differential count revealed 68 per cent. polymorphonuclear cells. There were 4,500,000 red cells and the hemoglobin was 90 per cent.

Orientation, remote and recent memory, retention, calculation, general information, and test judgment were all good. He was in contact with his environment and understood the nature of the examination. Most of the time he was quiet and cooperative. At times he paced the floor and muttered vengefully, "The G— d— white-haired s— of a b—." He was extremely suspicious and on his guard. He stated that he had been worried and nervous because the business was lost and the taxes were going to take the house. He regarded the whole examination as "bunk," and "supposed it was all research stuff." When questioned about the Jewess with whom he had been acquainted he said, "So its all coming out now. How did you find out about that?" He would not discuss it further.

CASE 3.—Katherine C., aged 59, had been a widow for fifteen years and had had no children. She had been fairly active socially. About sixteen years ago, after the death of a brother, she had a period of nervousness and depression from which she recovered in about three months. Frank and George went to live with her about seventeen years ago, two years before the death of her husband. The three had lived together since, except for two years following George's marriage.

She began to show evidence of mental disturbance about the same time as Frank. Her memory began to fail; she ate little and did not sleep well; she talked about her brothers being ruined and the house being sold for taxes. She thought her sister-in-law was the cause of the ruin and that she had obtained possession of the house. She had an idea that all of them were to be sent to a federal prison because they owed a broom-man \$2.50. She blamed herself for not having given her brothers more financial aid. Twice she tied a rope around her neck with the intent to commit suicide. She became much incensed at her sister-in-law and pulled her hair and would not let go until they were forcibly separated. She walked the floor constantly and refused to eat for several days before being brought to the hospital.

*Examination.*—Physical examination showed senile changes. Many teeth were missing. The blood pressure was 140 systolic and 90 diastolic. The neurologic examination was negative. The urine was free from albumin, sugar and casts. The hemoglobin was 90 per cent. and there were 4,200,000 red blood cells per c.mm. There were 7,200 leukocytes of which 75 per cent. were polymorphonuclears and 21 per cent. mononuclear cells. Blood and spinal fluid Wassermann reactions were negative. The spinal fluid was under low pressure, contained 2 lymphocytes per c.mm., and the Pandy reaction was negative.

She was disoriented for time, and thought that she was in jail. Memory for remote and recent events, general information, calculation and test judgment were all poor. It was difficult to apply the tests because the patient did not cooperate well. She wrung her hands and then raised them to her face in an attitude of despair. When not being examined, she wandered aimlessly about the ward wringing her hands. She was in poor contact, and had no idea what the examination was about. She said she was very unhappy and her whole appearance was one of deep dejection. She repeated, "Oh dear, oh dear, what will I do? When I go out I will have to walk up and down the street." There was no spontaneous stream of talk. There was retardation in answering questions. She seemed to have difficulty in fixing her attention. She thought her brothers had been put in jail because of failure in business. She did not know their ages. She refused to talk about her sister-in-law, but put her head down on her hands as if exhausted and cried.

#### COMMENT

In the case of George, the diagnosis presented little difficulty. There was a history of a previous attack with recovery. The diagnosis made was manic-depressive depression with agitation. There was some improvement during the following three weeks.

In the case of Frank the diagnosis made was depression. The psychosis was not so deep, though at times the agitation was far greater. Recovery began within a week after admission to the hospital, though, even at the time of commitment to the state hospital three weeks later, he still had the idea that everything was hopelessly lost. While George was deeply self-accusatory, Frank was not, but showed a marked paranoid trend against his sister-in-law. Before entrance to the hospital he had neglected his appearance and allowed his beard to grow. After being in the hospital a short time he allowed himself to be shaved and was much more particular about the care of his clothes.

The sister, Katherine, was deeply dejected and greatly agitated during her entire stay in the Psychopathic Hospital. There was also an indefinite history of a preceding attack in her case. Without further observation, it was impossible to exclude the factor of senility.

All three patients were committed to a state hospital.

In discussing contagious insanity Chagnon<sup>1</sup> says that according to Falret and Lasègue, in 1873, certain very special conditions must be present; the same family life; the same sentiments; and the persons must be apart from other influences. One must play the active rôle, the others are passive. Chagnon observed a case of *folie à trois*. Three dressmakers, aged 24, 27 and 33 years respectively, lived together. The family history had no important bearings. All were unmarried; their affections were concentrated on a pet dog; the death of this animal from eating bad meat completely upset them. The three

1. Chagnon: *Union méd. du Canada* 29:597, 1900.

sisters, after eating some meat, presented symptoms of intoxication and their imagination became filled with the obsession that they were poisoned like the dog; this was especially fostered by one sister. She imagined that a neighbor poisoned the dog in order to enter the apartment to rob it and to poison them. All became delirious.

Fillassier<sup>2</sup> reported a case of a woman, aged 37, who was active in communicating the condition to her brother and to his weak-minded mistress. Delusions of persecution with illusions were present. He describes this as: "A common case of mental contagion which implies the existence of a passive subject open to suggestion and an active subject who exercises this influence. There is always a 'contagioner' and a 'contagionee,' but neither is conscious that the influence is being exerted."

Griffin<sup>3</sup> reported the case of two sisters (M. and T.) who went to see another married sister (C.) who was suffering from mental aberration. They nursed this sister for a week by day and night, lying down on the sick sister's bed during the night so that they had scarcely any sleep or rest. One sister (M.) appeared to be all right when leaving, but developed symptoms of insanity the next day. The other sister (T.) became mentally affected on the day before leaving and had to be taken home by her husband. The insanity in the case of Sister C. consisted of restlessness, delusions and hallucinations of sight and hearing. Griffin reported this as a striking example of persons becoming insane from companionship, not in consequence of direct transference of morbid ideas, but from the shock caused by witnessing the illness and the strain of nursing the patient. The communicated insanity was identical with that in the original patient. The three sisters were greatly attached to each other. All three recovered. There was no hereditary taint.

Pages<sup>4</sup> reported a case of *délire à trois* in which the same type of insanity affected a mother and two daughters. At first they were the victims of imaginary persecutions and robbery; later they suffered from megalomania. The younger daughter was the active element and the most severely affected. Her sister improved and the mother recovered. Pages said that, in communicated insanity, a hereditary history is not necessary; but a nervous predisposition with weakness of morality or intellectual will power are necessary. The communicated mental disorder is at first limited in extent and embraces only one or two fixed ideas. In the case reported, however, heredity, was a factor; the father was old and the mother nervous.

2. Fillassier: *Rev. de psychiat.* **13**:466 (Aug.) 1909.

3. Griffin: *J. Ment. Sci.* **46**:3, 1900.

4. Pages: *Délire à trois*. *Ann. méd. psychol.* **8**:371, 1888.

Halberstad<sup>5</sup> said that simultaneous insanity must be differentiated from insanity from contagion in estimating the value of some etiologic factor. He thinks that inheritance of the psychosis, which is relatively frequent, explains the identical evolution of the psychic symptoms in the members of the same family.

Halberstad divides cases in which direct contagion is probable, into two categories: (1) One psychosis provokes another without resemblance to it; (2) one psychosis provokes another similar both as to symptoms and evolution, but of different intensity. Among true cases of insanity by contagion, the author distinguishes a special group, viz., a psychosis on the basis of ideas of reference, but with weak systematization and slow evolution, with generally a weak mentality.

Dercum and Mills,<sup>6</sup> in 1887, reported three negroes, two daughters and their mother, who all became victims of acute mania communicated by one daughter who had become violently maniacal under the influence of a "medium" (spiritualist), a white woman whom she had consulted and who had told her that she was possessed by an evil spirit. Puero<sup>7</sup> reported the case of a girl with systematized delusions of persecution who communicated the mental state to three sisters and two brothers. Each reacted according to his intelligence. The weakest was a deafmute aged 42, who became the victim of numerous hallucinations.

Martineng,<sup>8</sup> in 1887, reported a case of communicated insanity in a family consisting of father, mother, two sons and three daughters. Three children were married and living away from home; two daughters, aged 32 and 29 respectively, lived with their parents. The character of the younger daughter suddenly changed and she became the victim of a mystical delirium with ideas of religious persecution and hallucinations. The elder sister became affected in the same way. The first sister was sent to an asylum; the second then recovered. The first sister returned home and remained normal for fourteen months; then there suddenly occurred a new outburst of insanity and the sister, father and mother successively became affected in the same way. All had been completely exhausted by watching and nursing the youngest sister. The young daughter was again sent to the asylum and the other members of the family recovered. This was a case of hysterical insanity characterized by manic agitation, convulsive nervous phenomena, delusions of persecution and hallucinations of hearing, etc. There was an unequal distribution of these symptoms in the persons to whom the insanity was communicated.

5. Halberstad: Thèse de Paris, 1906.

6. Dercum, F. X., and Mills, C. K.: *J. Nerv. & Ment. Dis.* **14**:594, 1887.

7. Puero: *Riv. frenopat. espan.*, 1911; abstracted in *Encéphale* **7**:197, 1912.

8. Martineng: *Ann. méd. psychol.* **6**:383, 1887.

Woods,<sup>9</sup> in 1897, reported three cases of entire families (one consisting of father, mother, son and daughter; the second of five persons, a father, one son and three daughters; the third, a mother, son and two daughters) becoming simultaneously insane. The insanity was of acute hysterical type and arose from direct communication with a member of the family already insane. In two cases there was hereditary predisposition and in almost all a scrofulous and neurotic tendency. In the first two the author says that shock acting on constitutions already enfeebled appears to have been the exciting cause. All cases were of the same type, i. e., highly neurotic persons living in remote districts with little to divert their thoughts from the surroundings; when anxiety arose they were unable to bear the strain.

Archambault<sup>10</sup> called attention to a case in which a woman with delusions of persecution communicated her insanity to her father and her daughter. A case of persecutory hallucinosis communicated to seven members of a family by the eldest daughter is reported by Etchepare.<sup>11</sup> Elements of the contagion were found in the heredity, education and common life. Levassort<sup>12</sup> reported the case of a woman who caused her husband to be murdered by their eldest son. The woman and her four children, who were all perverts with hereditary taints, had for months planned a method of killing the father. There was a complete absence of normal sentiment and feeling of responsibility. The author termed this condition "familial moral insanity."

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9. Woods: *J. Ment. Sci.* **43**:822, 1897.

10. Archambault: *Le petit indépendant méd.*, May, 1907; abstracted in *Encéphale* **3**:159, 1908.

11. Etchepare. *Ann. méd. psychol.* **11**:1, 1910.

12. Levassort: *Encéphale* **7**:485, 1912.

## Critical Reviews

### THE PATHOLOGY OF MOTOR PARALYSIS BY LEAD\*

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- I. INTRODUCTION.
- II. THE TERM "NEURONITIS."
- III. THE CLINICAL ASPECT OF LEAD NEURITIS.
- IV. REVIEW OF THE LITERATURE DEALING WITH PATHOLOGY.
  - 1. Pathology of the Muscles.
  - 2. Pathology of the Nerves.
  - 3. Pathology of the Roots.
  - 4. Pathology of the Spinal Cord.
- V. CONCLUSIONS.

#### I. INTRODUCTION

Discussion of the site of attack, whether peripheral, central or both, in diseases of the peripheral neurons, is still active. A recent reference<sup>1</sup> showing the present attitude in England toward the whole problem of multiple neuritis follows:

Peripheral neuritis, multiple neuritis and polyneuritis are all terms applied to a complex of symptoms due to disease affecting the peripheral motor and sensory neurons. The disease as a whole is characterized by the facts that many nerves are affected at the same time or in rapid succession, that this affection of the nerve is always bilateral and usually symmetrical and that the more distal parts of the neurons are especially attacked, producing symptoms largely limited to the distal parts of the limbs. *It is important, however, to realize that the conception of the disease as one limited to the peripheral nerves is erroneous* [italics ours]. As will be pointed out, the action of the infection or intoxication falls, in many cases at all events, on the central as well as the peripheral nervous system.

\*Read at the meeting of the Section of Neurology and Psychiatry of the New York Academy of Medicine, April 10, 1923.

1. Feiling: Multiple Neuritis, in Oxford Loose Leaf System of Medicine 6:647, 1921.

Barnes,<sup>2</sup> in 1902, described as "toxic degeneration of the lower motor neurone" several cases which, in certain respects, conformed clinically to the condition called multiple neuritis. Holmes,<sup>3</sup> in 1917, and Bashford, Bradford and Wilson,<sup>4</sup> in 1918, established that in a certain type of infectious polyneuritis the anterior horn cells were involved early and that the changes in the nerve trunks were patchy and both parenchymatous and exudative in type. Walshe,<sup>5</sup> in 1918, demonstrated that in certain cases of localized paralysis which were associated with cutaneous diphtheritic lesions, the infection was carried through the lymphatic channels along the nerve trunk and that symptoms appeared when the infection reached the anterior horn cells. In these cases the nerves themselves were free from pathologic changes. In the widespread forms of diphtheritic paralysis in which the toxins are generalized, the nervous system is involved both centrally and peripherally—that is, both the anterior horn cells and the peripheral nerve trunks are affected. These writers have fairly well established that the pathologic process, in some forms of so-called multiple neuritis, involves the entire lower neuron.

Oppenheim,<sup>6</sup> in his textbook (edition of 1911), suggests that in arsenical paralysis the lesions are both central and peripheral, and thus it would be fair to interpret his statements as meaning that the entire lower neuron is involved in this toxic form of multiple neuritis. Kraus,<sup>7</sup> in a review of the literature of diabetic neuritis, found that: "Satisfactory clinical evidence of primary extramedullary involvement of the peripheral neurons, that is, peripheral neuritis, has not been obtained, either from a review of the clinical and pathologic reports in the literature or from my own experience."

## II. THE TERM "NEURONITIS"

To combat the doctrine that the condition called "multiple neuritis" is a peripheral neuritis, is no easy task. Not only must the past literature be reviewed, but experimental, clinical and pathologic studies must be made anew. And, even when these are accomplished, one

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2. Barnes: Toxic Degeneration of the Lower Motor Neurone Simulating Peripheral Neuritis, *Brain* **25**:479, 1902.

3. Holmes, Gordon: Acute Febrile Polyneuritis, *Brit. M. J.* **2**:37, 1917.

4. Bradford, Bashford, and Wilson: Acute Infective Polyneuritis, *Brit. Quart. J. M.* **12**:88, 1918.

5. Walshe, F. M. R.: On the Pathogenesis of Diphtheritic Paralysis, *Brit. Quart. J. M.* **11**:191, 1918; Post Diphtheritic Paralysis, *Lancet* **2**:232 (Aug. 24) 1918.

6. Oppenheim, H.: Textbook of Nervous Diseases, Otto Schulze & Co., Edinburgh: 1911, Vol. 1, p. 530.

7. Kraus, W. M.: Involvement of the Peripheral Neurons in Diabetes Mellitus, *Arch. Neurol. & Psychiat.* **7**:208 (Feb.) 1922.

still has to face the inertia due to the ingrained concept that "multiple neuritis" is a peripheral neuritis—that the lesions are primarily or exclusively (or both) in the peripheral twigs of the nerves.

A satisfactory title for this involvement of peripheral neurons has interested several writers. The term neuronitis, so far as we have been able to find, was first used in this country by Charles K. Mills,<sup>8</sup> in 1898, when he wrote: "With regard to multiple neuritis it is no new thing for me to teach the involvement of both nerve and cord in a large number, if not in a majority, of the cases. Long before the theory of the neuron was thought of, I expressed my belief that in many cases of so-called multiple neuritis both periphery and spinal centers were implicated; in other words, as then expressed, we had a concurrence of multiple neuritis and of generalized poliomyelitis in the same case. . . . These diseases are, therefore, in some cases at least, neither instances of neuritis in a strict sense or of poliomyelitis. They are, perhaps, what might be termed 'neuronitis,' and this term has been suggested, but seems to have an unnatural sound even to a neurological ear."

In 1907, E. Farquhar Buzzard,<sup>9</sup> when speaking of acute toxic polyneuritis, said: "The question of title again presents itself, and there is a general feeling that this group might well have a distinctive name, if only a suitable one could be found. 'Toxic degeneration of the lower motor neuron' is too long, and 'motor neuronitis' is open to popular, if not medicolegal, misinterpretation." In 1919, Foster Kennedy,<sup>10</sup> in describing certain cases forming a subgroup of epidemic encephalitis, labeled them "infective neuronitis." The term "neuronitis" is, therefore, not new, and, though possibly not generally used, has at least come to have a sound to the "neurological ear" which can no longer be designated as "unnatural."

After all, what is desirable is that clinical pictures be described and labeled precisely so as to indicate, as far as possible, their cause, pathology and course. We do not wish to give the impression that we maintain that all conditions now spoken of as neuritis, are neuronitis. But three forms of so-called multiple neuritis—due to certain infections and toxins—are affections of the entire lower motor neuron. In diabetes the intramedullary portions of neurons are probably the primary site of attack.<sup>7</sup>

8. Mills, C. K.: The Reclassification of Some Organic Nervous Diseases on the Basis of the Neuron, *J. A. M. A.* **31**:11 (July 2) 1898.

9. Buzzard, E. F.: On Certain Acute Infective or Toxic Conditions of the Nervous System, *Brain* **30**:85, 1907.

10. Kennedy, Foster: Infective Neuronitis, *Arch. Neurol. & Psychiat.* **2**:621 (Dec.) 1919.

This critical review aims to show that the same is true in lead paralysis, but refers only to that portion of the literature that gives facts of pathology. No mention is made of references dealing with the subject from the clinical standpoint or in which expressions of opinion as to pathology are based only on clinical evidence.

### III. THE CLINICAL ASPECT OF LEAD NEURITIS

Lead paralysis may be acute or chronic, and may appear without preceding evidence of lead poisoning. It usually develops a few weeks after such prodromata as colic and pains in the joints and muscles. Once developed, the involvement may seem to be limited entirely to the lower motor neuron. In adults, the paralysis is commonly found only in the upper extremities; in children, the legs are usually first affected. The paralysis is not always bilateral, much less symmetrical.

In the upper extremities there are three principal types of paralysis: upper arm, lower arm, and Aran-Duchenne type. In the last there is extensive atrophy of the intrinsic muscles of the hand, often without commensurate weakness. As a rule, lead paralysis affects the lower arm muscles supplied by the seventh and eighth cervical, and the first thoracic segments. The preference for muscles supplied by segments of the cord rather than for those supplied by a particular nerve or nerves, gives rise to the query—Why is lead paralysis called a peripheral neuritis? The supinator longus is supplied by fibers from the sixth cervical segment. If, in a musculospiral paralysis, the supinator escapes and the sixth anterior root is normal, there is reason for suspecting that the primary change is in the cord. It would be remarkable for the process to begin in the distal parts of a nerve (in this case the musculospiral), extend upward to the cord and yet not involve all the roots of the nerve. Several writers point out that when the supinator longus is involved in lead paralysis, the biceps and deltoid, which are supplied by the fifth and sixth cervical segments, are also affected. This fact leads to the conclusion that the origin is not peripheral. In addition, there are now a number of reports of cases with the clinical picture of amyotrophic lateral sclerosis. The central motor neuron is, therefore, affected in some cases. Potain<sup>11</sup> (1887) pointed out that the dorsal interosseous artery alone supplies the muscles usually involved in lead paralysis, and suggested that general vascular changes, due to lead poisoning, would first show themselves in these muscles because their supply of blood is poor. In this way he provides a possible peripheral origin for palsy of muscles supplied by particular spinal segments. The explanation is ingenious, but is scarcely acceptable in view of the large number of nervous system findings.

11. Potain: Sur un cas de paralysie hystéro-saturnine, *Bull. méd.* 1:851, 1887.

The disproportion between atrophy and weakness of the intrinsic muscles of the hand in the Aran-Duchenne type of lead paralysis should not be passed by without comment. In curare poisoning the motor end plates are attacked and there is complete loss of the transmission of voluntary motor impulses. In family periodic paralysis it has been suggested that involvement of the motor end plates is responsible for the clinical picture. In traumatic lesions of the peripheral nerve trunks the weakness and atrophy are more nearly commensurate. In recognized forms of muscular atrophy due to chronic and gradual degeneration of the anterior horns, we usually find that the atrophy is greater than the weakness. These facts certainly suggest that the Aran-Duchenne type of lead palsy represents a poliomyelitic rather than a terminal neuritic process.

#### IV. A REVIEW OF THE LITERATURE DEALING WITH PATHOLOGY

An examination of the spinal cord, the roots, the mixed nerve trunks, the muscles and the blood vessels that supply each of these parts is necessary in every case of lead paralysis to permit a trustworthy conclusion as to the primary causal pathologic process. The scarcity of such reports in the literature of the subject is striking. Again, it is usual to find no mention of the stains used in preparing the tissues. If the Marchi or Weigert stains bring out pathologic changes, it is safe to assume their existence. However, the converse is by no means true. Not until Nissl's work,<sup>12</sup> in 1892, was there a method of demonstrating finer cellular pathology. Hence, many of the older reports must be disregarded when they deny the existence of alterations in the spinal cord. In the great majority of cases reported, there was no examination of the anterior and posterior spinal roots. This omission is all important.

Nerve trunks may show degeneration of moderate degree, and in a given case the cord may show slight or no alteration. But if the posterior roots are found to be normal and changes are present only in the anterior roots; and if some roots only of the segments that enter into the makeup of a nerve trunk are involved, then we have important evidence that the degeneration is due to altered cord structure. Another failure in many reports is lack of attention to the condition of the blood vessels of the cord and nerves; this is particularly important because lead is a general poison that is especially apt to produce arteriosclerosis. Feiling<sup>1</sup> states: "In some cases, indeed, other systems of the body,

12. Nissl: Ueber experimentelle erzeugte Veränderungen an den Vorderhornzellen des Rückenmarks bei Kaninchen mit Demonstration mikroskopischen Präparate, *Allg. Zeitschr. f. Psychiat.* 48:675, 1892; *Versammlung des psychiat. Vereins der Rheinprovinz in Bonn*: Nov. 14, 1891.

especially the heart, are equally affected." Finally, if a patient with lead paralysis is found to have also either syphilis, tuberculosis, diabetes mellitus or chronic alcoholism, examinations of his tissues cannot be relied on for information concerning the pathology of this disease.

#### 1. PATHOLOGY OF THE MUSCLES

Many authors have found the muscles involved. The common pathological picture is proliferation of connective tissue and atrophy of muscle fibers. Debove and Renaut<sup>13</sup> (1876) raised the question whether section of a nerve would produce the same changes. Friedlander<sup>14</sup> (1879) stated that he believed lead paralysis to be a myopathy. Although later writers<sup>15</sup> have reported cases of lead paralysis resembling progressive muscular atrophy, Harnack<sup>16</sup> (1878), Friedlander<sup>14</sup> (1879), and Kast<sup>17</sup> (1880) were the only authors to maintain that the primary pathologic change occurs in the muscles. More recent investigations indicate that this view is incorrect.

*Summary.*—There is no adequate evidence that the primary pathologic changes are in the muscles.

#### 2. PATHOLOGY OF THE NERVES

The process is described as a parenchymatous degeneration, beginning usually in the distal parts of the nerve or nerves involved. Goadby and Goodbody<sup>18</sup> (1909), found hemorrhages in the nerves and muscles of poisoned rabbits, but no changes in the cord. Moreover, they found no degeneration of the nerves. We cannot interpret these findings because they are unique; the experiments need to be repeated. Stieglitz<sup>19</sup> (1892), working on dogs, found the nerves often less affected than the cord. Other writers held that in man the degeneration of nerves is usually more intense than that of the cord. Dejerine<sup>20</sup>

13. Debove and Renaut: Note sur les lésions des faisceaux primitifs des muscles volontaires dans l'atrophie musculaire progressive et dans la paralysie saturnine, *Gaz. méd. de Paris*, Series 4 5:114, 1876.

14. Friedlander: Anatomische Untersuchung eines Falles von Bleilähmung nebst Begründung der myopathischen Natur dieser Affektion, *Virchow's Arch.* 75:24, 1879.

15. Grinker, Julius: Multiple Neuritis Simulating Progressive Muscular Atrophy, *J. A. M. A.* 49:830 (March 9) 1907.

16. Harnack: Die Wirkungen des Blei auf den thierischen Organismus, *Arch. f. exp. Pathol. u. Pharm.* 9:152, 1878.

17. Kast: Notigen zur Bleilähmung, *Centralbl. f. Nervenheilk.* 3:137, 1880.

18. Goadby and Goodbody: A Note on the Pathology of Lead Poisoning, *Lancet*, 2:988 (Oct. 2) 1909.

19. Stieglitz, L.: Eine experimentelle Untersuchung über Bleivergiftung mit besonderer Berücksichtigung der Veränderungen am Nervensystem, *Arch. f. Psychiat.* 24:1, 1892.

20. Dejerine, J.: Recherches sur les lésions du système nerveux dans la paralysie saturnine, *Compt. rend. Soc. de biol.*, Series 7 1:11, 1879.

(1879) raised the question whether lead paralysis is due to an ascending or descending degeneration, and quoted Ranvier as denying the possibility of an ascending neuritis. Popow<sup>21</sup> (1883) stated that Lancereaux<sup>22</sup> (1871), Gombault<sup>23</sup> (1873), Bernhardt<sup>24</sup> (1874), Minor<sup>25</sup> (1877), Eisenlohr<sup>26</sup> (1879), Mayor<sup>27</sup> (1877), and Westphal<sup>28</sup> (1874) incline to the opinion that the pathology is primarily in the peripheral nerves, while Remak<sup>29</sup> (1875), Erb<sup>30</sup> (1874, 1883), Renault<sup>31</sup> (1878), and Bernhardt<sup>24, 32</sup> (1874, 1878) on clinical grounds, regarded the lesions as primarily spinal.

These differences of opinion reflect the spirited controversy existing in the early decades of the development of neurology, and also reflect the difference of opinion concerning the site of pathologic changes underlying the clinical condition known as multiple neuritis. In other words, some were inclined to believe the condition neuritis, while others considered it a neuronitis (since involvement of the anterior horn would imply a subsequent involvement of the peripheral motor fibers).

Of the cases cited by Popow<sup>21</sup> (1883), several must be discounted. In the first place, tissue staining at that time was not delicate enough to

21. Popow, N.: Ueber die Veränderungen im Rückenmark nach Vergiftungen mit Arsen, Blei und Quecksilber, *Virchow's Arch.* **93**:351, 1883.

22. Lancereaux: Saturnisme chronique avec accès de goutte et arthrites uratiques, *Gaz. méd. de Paris*, **26**:385, 1871.

23. Gombault: Contribution à l'histoire anatomique de l'atrophie musculaire saturnine, *Arch. de physiol.* **5**:592, 1873.

24. Bernhardt: Zur Pathologie der Radialisparalyse, *Arch. f. Psychiat.* **5**:601, 1874.

25. Minor, L. S.: On Lead Paralysis, *Voyenno M. J.*, St. Petersburg, **141**:149, 1887.

26. Eisenlohr: Idiopathische subakute Muskellähmung und Atrophie, *Centralb. f. Nervenheilk.* **2**:100, 1879; Ueber einige Lähmungsformen spinalen und peripheren Ursprunges, *Arch. f. klin. Med.* **26**:642, 1880.

27. Mayor: Lésions des nerfs intramusculaires dans un cas de paralysie saturnine, *Gaz. méd. No. 12. Compt. rend. Soc. de biol.*, 1877, Paris, 1879, 6th series, Vol. 4, p. 213.

28. Westphal: Ueber eine Veränderung des N. radialis bei Bleilähmung, *Arch. f. Psychiat.* **5**:776, 1874.

29. Remak, E.: Zur Pathogenese der Bleilähmung, *Arch. f. Psychiat.* **6**:1, 1875.

30. Erb, Wilhelm: Ein Fall von Bleilähmung, *Arch. f. Psychiat.* **5**:445, 1874; Bemerkungen über gewisse Formen der neurotischen Atrophie, *Neurol. Centralbl.* **2**:481, 1883.

31. Renault: Remarques anatomiques et cliniques sur deux points particuliers de l'intoxication saturnine chronique, *Gaz. méd. de Paris, Series 5* **7**:394, 1878.

32. Bernhardt, M.: Ueber Bleilähmung und Subacute Atrophische Spinal-lähmung Erwachsener, *Berlin Klin. Woch.* **18**:273, 1878.

show finer cord changes. Eisenlohr's patient<sup>20</sup> (1879) was tuberculous and a chronic alcoholic. Gombault's<sup>33</sup> (1880) reports were not complete. Bernhardt<sup>32</sup> (1878) found cord changes in the cervical region. Westphal's<sup>28</sup> case (1874) was not completely examined. Popow<sup>21</sup> (1883), after quoting a number of others who believed that the primary pathology was in the peripheral nerves, himself concludes that the pathologic changes are primarily in the cord. Other later writers continued the discussion without contributing any new pathologic facts to support their theories.

In 1911, Anglada<sup>34</sup> reviewed the literature and came to the conclusion that lead paralysis is a peripheral neuritis. Others hold that while the nerves are first involved the cord is attacked later. Zunker<sup>35</sup> (1880) and Wilson<sup>36</sup> (1907) are in this group. The latter reports Marie<sup>37</sup> (1904) as follows: "An exception can be made in some forms of muscular atrophy due to lead poisoning. These forms show clearly defined lesions of the anterior horn cells in the gray matter (Von Monakow, Oeller, Oppenheim), and may be grouped with chronic anterior poliomyelitis. Wilson then states: "It may be taken, then, that the muscular atrophy of lead poisoning is, in part at least, attributable to the action of the toxic agent on the motor cells of the cord."

The majority of recent writers find changes both in nerves and cord and reach one of two conclusions: (1) The paralysis is primarily of cord origin; (2) it may begin either in the nerves or in the cord. Lead, according to some, acts primarily on the nerve tissue; according to others, the lead affects the blood vessels (Vierordt,<sup>38</sup> 1887) which, in turn, by becoming diseased produce changes in the nerve tissue.

*Summary.*—On the whole, then, the case for a solely peripheral nerve origin of lead paralysis is weak. The term neuritis tells but a part of the story.

33. Gombault: Note sur l'état des nerfs périphériques dans l'empoisonnement lent par le plomb chez le cochon d'Inde, *Prog. méd.* **8**:181, 1880; Contribution à l'étude anatomique de la névrite parenchymateuse subaiguë et chronique, *Arch. d. neurol.* **1**:11 and 177, 1880 and 1881.

34. Anglada, J.: Paralyse saturnine généralisée probablement polynévritique, *Soc. des sc. méd. Montpellier méd.*, **32**:427 (April 30) 1911; abstracted in *Rev. neurol.* **23**:264, 1912.

35. Zunker, E.: Zur Pathologie der Bleilähmung, *Zeitschr. f. klin. Med.* **1**:496, 1880.

36. Wilson, S. A. K.: The Amyotrophy of Chronic Lead Poisoning: Amyotrophic Lateral Sclerosis of Toxic Origin, *Rev. Neurol. & Psychiat.* **5**:441, 1907.

37. Marie, Pierre: Affections médullaires par intoxications, *Traité de méd.*—Masson et Cie, Ed. 2, Paris: 1904, **9**:599.

38. Vierordt: Zur Frage vom Wesen der Bleilähmung, *Arch. f. Psychiat.* **18**:48, 1887.

## 3. PATHOLOGY OF THE ROOTS

Fisher<sup>39</sup> (1892), Laslett and Warrington<sup>40</sup> (1898), Philippe and Gothard<sup>41</sup> (1903), found changes in the anterior roots only. Stieglitz<sup>19</sup> (1892) and Spiller<sup>42</sup> (1903) found the posterior roots and ganglions also affected. Popow<sup>21</sup> (1883) and Spiller and Longcope<sup>43</sup> (1906), found the roots normal. De Watteville<sup>44</sup> (1880) concluded that the primary lesion is in either the cord or the roots. Ceni<sup>45</sup> (1897) found changes in the cord, roots and muscles. These are the only writers who describe root changes, accompanied as well by changes in the nerves and cord. The presence of root changes by no means indicates that the origin of lead paralysis is in the roots. But such findings are of great importance in determining whether the primary change is in the nerves or the cord.

*Summary.*—Insufficient attention has been paid to the condition of the roots to warrant definite conclusions.

## 4. PATHOLOGY OF THE SPINAL CORD

Changes may be present in the meninges, the blood vessels, the gray or the white matter of the cord. The gray matter and vessels commonly show some alteration. Bernhardt<sup>24</sup> (1874), Kussmaul and Maier<sup>46</sup> (1872), Fisher<sup>39</sup> (1892), Stieglitz<sup>19</sup> (1892), Nissl<sup>12</sup> (1892), all remark on vascular congestion or the presence of capillary hemorrhages. Schaffer<sup>47</sup> (1893) found changes in the cords of dogs. Thickening of the meninges is described by Bernhardt<sup>24</sup> (1874) and by Fisher<sup>39</sup> (1892). The majority of writers do not comment on these two points. Mosny and Harvier<sup>48</sup> (1908) found meningeal involve-

39. Fisher, E. D.: Lead Poisoning with Special Reference to the Spinal Cord and Peripheral Nerve Lesions, *Am. J. Med. Sc.* **104**:51, 1892.

40. Laslett and Warrington: The Morbid Anatomy of a Case of Lead Paralysis, *Brain* **21**:224, 1898.

41. Philippe and Gothard: Contribution à l'étude de l'origine centrale de la paralysie saturnine, *Rev. neurol.* **11**:117, 1903.

42. Spiller, W. G.: The Pathological Changes in the Nervous System in a Case of Lead Poisoning, *J. Med. Res.* **5**:142, 1903.

43. Spiller and Longcope: Multiple Motor Neuritis, Including Landry's Paralysis and Lead Palsy, *New York Med. Rec.* **70**:81 (July 21) 1906.

44. De Watteville: On the Pathogeny of Lead Palsy, *Lancet* **2**:44 (July 10) 1880.

45. Ceni: Sur la pathologie de la paralysie saturnine, *Arch. f. Psychiat.* **29**:566, 1897.

46. Kussmaul, A., and Maier, R.: Zur pathologischen Anatomie des chronischen Saturnismus, *Deutsch. Arch. f. klin. Med.* **9**:285, 1872.

47. Schaffer: Ueber die Veränderungen der Nervenzellen bei experimenteller chronischer Blei Arsen und Antimon-Vergiftungen, *Ungar. Arch. f. Med.* **2**:43, 1893.

48. Mosny and Harvier: L'amaurose saturnine, *Trib. méd.* **46**: 1908.

ment with lymphocytes in the cerebrospinal fluid. Degenerative processes in the anterior horns, most intense in the lower cervical region, were found by Bernhardt<sup>24</sup> (1874), Vulpian<sup>49</sup> (1879), Von Monakow<sup>50</sup> (1880), Zunker<sup>55</sup> (1880), Oeller<sup>51</sup> (1883), Oppenheim<sup>52</sup> (1885), Schultze<sup>53</sup> (1885), Oppenheim and Siemerling<sup>54</sup> (1887), Eichorst<sup>55</sup> (1890), Nissl<sup>12</sup> (1892), Stieglitz<sup>19</sup> (1892), Laslett and Warrington<sup>40</sup> (1898), Spiller<sup>42</sup> (1903), Bernard and Salomon<sup>56</sup> (1903), Philippe and Gothard<sup>41</sup> (1903), Spiller and Longcope<sup>43</sup> (1906) and Claude and Loyez<sup>57</sup> (1914). Of these, Zunker (pathologic findings) and Oeller (nephritis case) have been criticised by Spiller. Birdsall<sup>58</sup> (1882) reported a case in which he considered that cord changes were present. But in the discussion that followed, E. C. Spitzka disagreed on that point and Putnam-Jacoby suggested that the increased vascularity of the affected parts might indicate slackened circulation which could lead to impaired nutrition of the nerve tissues. This case must be disregarded on the score of difference of opinion among those who examined the tissues—even if, for the sake of argument, the staining technic was deemed sufficient to show finer pathology. Dreschfeld<sup>59</sup> (1885) reported the cord normal in his only necropsy. Ceni<sup>45</sup> (1897) found changes in the cord, roots and muscles.

Alterations in the white matter of the cord were noted by Fisher<sup>39</sup> (1892), Goldflam<sup>60</sup> (1893) and Eichorst<sup>55</sup> (1890).

*Summary.*—The commonest pathologic changes in the cord are in the vessels and the gray matter. The degree of involvement of the

49. Vulpian: *Leçons sur les maladies du système nerveux*, Paris **2**:158, 1879.

50. Von Monakow: *Zur pathologischen Anatomie der Bleilähmung und der saturninen Encephalopathie*, Arch. f. Psychiat. **10**:495, 1880.

51. Oeller: *Zur pathologischen Anatomie der Bleilähmung*, Munich: 1883.

52. Oppenheim, H.: *Zur pathologischen Anatomie der Bleilähmung*, Arch. f. Psychiat. **16**:476, 1885.

53. Schultze: *Ueber Bleilähmung*, Arch. f. Psychiat. **16**:791, 1885.

54. Oppenheim and Siemerling: *Beiträge zur pathologischen Anatomie der Tabes Dorsalis und der peripherischen Nervenerkrankung*, Arch. f. Psychiat. **18**:487, 1887.

55. Eichorst: *Beiträge zur Pathologie der Nerven und Muskeln*, Virchow's Arch. **120**:217, 1890.

56. Bernard and Salomon: *Un cas de paralysie saturnine à type radiculaire*, Rev. neurol. **11**:722, 1903.

57. Claude and Loyez: *Etude d'un cas d'intoxication saturnine avec paralysie et atrophies musculaires de date ancienne*, Encéphale **9**:30 (July) 1914; abstracted in Rev. neurol. **30**:288, 1914.

58. Birdsall: *A Contribution to the Pathological Anatomy of Lead Paralysis*, Am. J. Neurol. & Psychiat. **1**:176, 1882.

59. Dreschfeld: *On Alcoholic Paralysis*, Brain **8**:433, 1885.

60. Goldflam: *Ein Fall von Bleilähmung*, Deutsch. Zeitschr. f. Nervenheilk. **3**:343, 1893.

nerves and cord varies from case to case. Involvement of the white matter may, at times, produce pseudotabes and lateral sclerosis.

It is granted by most recent authors that pathologic changes exist in the spinal cord. Some writers hold that the changes are primary; some maintain that the anterior horn changes are a "réaction à distance"; others believe that in typical and early cases of lead paralysis the cord is not involved; and finally, some (e. g., Vierordt, 1887) suggest that, since lead is a general poison and produces marked arterial changes throughout the body, the site of election for the primary pathology will depend on individual factors in each case. Erb<sup>30</sup> (1883) and Remak<sup>61</sup> (1862) were the first to suggest that the pathology is primary in the cord, regardless of the possible absence of demonstrable cord changes. They considered that functional impairment of the cord produced degenerative processes in the distal parts of the peripheral nerves. Spiller<sup>42</sup> (1903) stated:

I conclude, therefore, from my study of this case and of the literature that lead affects the brain and its pia, the nerve cells of the anterior horns of the spinal cord, the ganglia on the posterior roots, the peripheral nerve fibers, and the muscles.

It seems impossible to determine whether its effects are first manifested in alteration of the peripheral motor fibers or of the motor cell bodies of the spinal cord, but inasmuch as *both peripheral nerve fibers and motor cell bodies are sooner or later affected* [italics our], this question is not a very important one.

The condition is, therefore, essentially a peripheral neuron disease or neuronitis.

#### V. CONCLUSIONS

In estimating the value of past contributions it seemed justifiable to eliminate from consideration case reports in which conspicuously incomplete pathologic examinations were made. Expressions of opinion based on clinical observations, without pathologic facts, have to some extent been discounted. Reports prior to Nissl's work (1892) that the spinal cord was normal, or in which the stains used are not mentioned, have been viewed with question.

In further investigations we feel that a study of root involvement is important. If it should be found that only a part of those entering into the make-up of a motor nerve trunk are affected, the obvious conclusion is that the primary change is central to the roots. Otherwise it would be necessary to explain how a disease process commencing in the distal parts of a motor nerve could extend centrad and fail to involve all the roots connected with that nerve.

It seems to us that the theory of "réaction à distance" does not account for the changes found in the anterior horn cells. The presence

61. Remak, E.: Ueber die Heilbarkeit der progressive Muskelatrophie, Oesterr. Zeitschr. f. prakt. Heilk. 2:35, 1862.

of pleocytosis in the spinal fluid and of pathologic changes in the meninges and white matter of the cord, accompanied by marked vascular congestion, features that have been reported by several authors, strongly favor the hypothesis that changes in the anterior horn cells are primary.<sup>62</sup>

For over fifty years there has been a controversy as to whether the origin of lead paralysis is neural or neuronal—whether the entire lower neuron or only its peripheral parts are affected. The trend of both facts and opinion in the last twenty years is in favor of the concept that the entire neuron is involved from the onset of symptoms.

In view of the changing conception of multiple neuritis consequent on the work of Holmes, Bradford and Bashford, Wilson and Walshe, it seems to us that lead paralysis, hitherto called a toxic form of multiple neuritis, should henceforth be regarded as a neuronitis.

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62. Additional references of interest are:

- Buzzard, Thomas: Two cases of Lead Paralysis, *Brain* **1**:121, 1878.  
Moritz, S.: A Contribution to the Pathological Anatomy of Lead Paralysis, *J. Anat. & Physiol.* **15**:78, 1880.  
Möbius: Ueber einige ungewöhnliche Fälle von Bleilähmung, *Centralbl. f. Nervenheilk.* **9**:6, 1886.  
Putnam: Lead Poisoning as a Cause of Muscular Incoordination, *Boston M. & S. J.* **113**:73 (July 28) 1887.  
Dejerine-Klumpke, T.: Des polynévrites en général, des paralysies et atrophies saturnines en particulier, Thèse de Paris, 1889.  
Jolly: Ueber Blei und Arseniklähmung, *Deutsch. med. Wchnschr.* **19**:97, 1893.  
Gowers, W. R.: Lead Palsy, *Clinical Lectures, Series 1*, London: 1895, p. 237.  
Remak, E.: Neuritis and Polyneuritis, Vienna: 1900.  
Villaret: Paralysies saturnines, *Gaz. d. hôp.* **76**:149 (Feb.) 1903.  
Gowers, W. R.: Metallic Poisoning, *Clinical Lectures, Series 2*, London: 1904, p. 140.  
Von Sarbo, A.: Spinale Muskelatrophie in Folge Bleivergiftung, *Deutsch. Zeitschr. f. Nervenheilk* **19**:249, 1909.  
Eichorst: Bleivergiftung und Rückenmarkskrankheiten, *Med. Klinik* **9**:201 (Feb. 9) 1913.  
Oliver, Thomas: Lead Poisoning, New York, Paul B. Hoeber, 1914, p. 147.

## Abstracts from Current Literature

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EXPERIMENTAL STUDIES ON MALIGNANT TUMORS OF THE CENTRAL NERVOUS SYSTEM. EDOUARD FLATAU, *Rev. neurol.* **28**: 987 (Sept.-Oct.) 1921.

This is a study of tumors produced experimentally in animals, for the purpose of comparing their genesis, evolution, and treatment with reference to malignant tumors in the human being, especially in the brain and spinal cord. It lays particular emphasis on the influence of the roentgen ray as a therapeutic measure. The number of animals experimented on totaled 1,061, of which 1,022 were white mice; the other 39 included rats, rabbits, dogs and monkeys. Most of the experiments involved the transplantation of cancerous adenoid tissue from one mouse to another.

The first section of the paper deals with the treatment of transplanted subcutaneous tumors. Flatau speaks of three methods: Physical, such as pressure; electricity, the roentgen ray; chemical, such as metal colloids, quinin, nitroglycerin, etc.; and biologic, including serum treatment, active and passive immunization, organotherapy, extirpation of internal glands, etc. In his studies relative to treatment of tumors of the central nervous system, he has felt it necessary to take into consideration methods employed in treating experimental subcutaneous tumors.

After enumerating at length the various agents used in treatment, the author describes his technic, or method of procedure. The first series of his experiments had to do with establishing *in vitro* the action of various chemical substances on sections of excised tumors. He considers as unique the fact that many chemical substances *in vitro* correct or totally abolish the generative power of neoplastic cells.

The second series of his experiments, he divides into two parts: First, experiments in which he transplanted (or "inoculated") the tumor, and immediately or shortly after injected certain substances; second, experiments in which he first injected substances into the animal, and then later transplanted the tumor, and then still later followed up the transplantation by further injection of the foreign substances. The results in both groups were negative. The substances injected had no perceptible effect on the growth of the tumors in mice, even when strongly toxic agents were used, such as arsenious acid, salts of silver, mercury, etc. The tumors grew steadily. Substances which killed the neoplastic cells *in vitro* had no effect on the tumor in the living organism. Such were the results obtained from studies of the subcutaneous tumors. From this, the author proceeds to experiments relative to tumors artificially produced in the central nervous system.

He has tried two methods of inducing such tumors: One by injection of an emulsion of the tumor substance which he wishes to reproduce; the other by transplanting a section or small portion of the original tumor. His experience has been that animals die almost instantaneously when the emulsion is injected into the brain. Cancers in mice rarely or never reproduce themselves in other animals. An emulsion of cancer from a dog injected into a monkey gives negative results.

The writer first considers experimental tumors of the cerebrum. He has discarded injection of emulsion in favor of transplantation of small sections. These artificially induced tumors develop rapidly as shown by necropsy. A week and a half to two weeks give maximum effects. By two weeks there are pronounced symptoms of general prostration, though definite hemiplegic signs are usually absent. Death usually follows in one to four weeks. The artificial growths may appear extradurally or intradurally and in the latter case often involve the deeper tissues. Inflammatory symptoms are never present.

Experiments on cerebellar tumors include those induced in the left or right lobe, in the median line, or by injection of emulsion into the meningeal culdesac. No symptoms appear earlier than the tenth to fourteenth day. At the end of that time a definite protuberance is noted, and then follow typical cerebellar symptoms, this picture being modified by movements peculiar to the animal, especially the attempt to balance itself on its head or nose with a rotary conical movement something like a slowly moving top. The animals die in from one to three weeks. Necropsy shows atrophy without evidence of inflammatory symptoms.

Experiments on the spinal cord give the picture of compression of a carcinomatous Pott's disease. The animals die in seventeen to twenty-one days.

Injections into the meningeal culdesac give diffuse lesions, sometimes in the cord, the cerebellopontile angle, or on the periphery of the cerebral hemispheres.

The author then discusses at length the influence of radium as a therapeutic measure. Preliminary to this he describes the effect of radium, employed for a period of from two to six hours over a number of days, on normal tissue. He especially speaks of the effect on the cerebral hemispheres, the occipital poles, and the quadrigeminal bodies. There is a notable degeneration of nerve cells in the cortex, sometimes invading the white matter and the vascular tissue, and a simultaneous appearance of neuroglia cells of the satellite type. The changes described conform rigorously to the areas exposed to radium.

After this discussion of experimentation on normal tissue, the writer describes the effects of radium on the artificially induced tumors. In the literature he finds practically no reference to the use of radium (up to the time his article appeared) as a therapeutic measure in brain or cord tumors. His own experiments on the cerebrum include two groups: first, those in which the inoculation and the use of radium are employed simultaneously; and second, cases in which there is a considerable lapse of time between the inoculation and the employment of radium. In the first series, the tumor never develops. In the second series, the efficacy of radium depends on the length of the interval between the inoculation and the first employment of radium. If radium is employed within one to two days after inoculations and repeated every two hours for a period of three days, the tumor does not develop. If the tumor is allowed to grow uninterruptedly for a longer period, the results are uncertain. In one case, however, the first in animal experimentation, the writer obtained a complete cure by radium without surgical operation after the tumor had been allowed to grow for two weeks. In the cerebellum he has not secured a complete cure in any case, though the development of the tumor has been arrested.

The paper concludes with a discussion of various methods of therapy to date in the matter of tumors of the central nervous system. The author

believes that radium thus far offers the best results. He brings out the important question as to why radium appears to exert a selective action on neoplastic cells rather than on the normal tissue, and leans to the idea that embryonic (neoplastic) tissue is less resistive. As for the biologic method, the production of antibodies, ferments, etc., as well as the use of endocrine substances, the author thinks that this field is still open. As compared with the use of radium the biologic approach is far from satisfactory.

It is still difficult to foresee what may yet be accomplished in the attempt to find a definitive solution for the treatment of neoplasms. It should be remembered that the paper deals only with experimentation on animals.

JONES, Detroit.

MIGRAINE IN CHILDREN. HANS CURSCHMANN, München, med. Wehnschr. 69:1747 (Dec. 22) 1922.

It is noteworthy that the text books on neurology, and even special monographs such as those of Möbius and Flatau, devote very little space to migraine in children. This is due partly to a lack of objective clinical observation, and to the fact that migraine is seen much more frequently in private practice than in clinics. It is also overlooked because it is not inquired into, and so much depends on a thorough history. It must be understood that migraine in older children, about the age of puberty, resembles that in the adult. The true migraine of childhood occurs in the school period up to the age of ten or twelve years. It has also been noted in suckling children.

In the interpretation of the paroxysmal manifestations, caution must be exercised in differentiating other nervous disturbances such as spasmophilia, which may be associated in a migrainous constitution. Curschmann has observed such conditions, for example, in a girl with bilateral headache, who when aged 4 or 5 years had her first attack of fainting, vomiting, exhaustion, drowsiness, and fever, all within twenty-four hours; these attacks occurred several times a year.

Associated abdominal symptoms are characteristic of migraine in children. These may vary from slight stomach ache to abdominal pains which more or less obliterate the headache and which may later even supplant the hemi-crania. Umbilical colics in infants and children are frequently of this type. The same is true of many cases resembling appendicitis, gastric ulcer, or duodenal disturbances. Migraine may also be hidden in vagotonic disturbances of the digestive organs and in spasmophilic abdominal equivalents. The author describes five cases of various grades of admixture of abdominal disturbances, including abdominal equivalents. In all cases the migraine was quite typical. The hereditary features were present and there was prompt restitutio ad integrum. In all cases there was nausea and vomiting in which the abdominal discomfort stood in the foreground. In one of these cases the abdominal attacks entirely replaced the headaches.

Along with migraine symptoms the author finds other symptoms of importance: heightened irritability of the vegetative nervous system; facial phenomena such as the Chvostek sign; spasmophilic reactions may occur, as also asthma, hiccup and an exudative diathesis. Fever associated with attacks is well known, and eosinophilia is mentioned.

Abdominal discomforts in migraine attacks, which also occur in adults, though less frequently, have many interpretations. It is the opinion of the author that these crises are splanchnogenic. Other organs and functions con-

trolled by the vegetative system may take part, at times in the form of excitement, at others in the form of inhibition, such as secretory and motor disturbances of the stomach, or changes in kidney function. Other vasomotor syndromes are also noted in the child, as those of cardiac origin. Two cases of this type are reported. Case 6, Mary R., aged 8, whose mother had a similar condition as a child, and whose grandmother had migraine, since the age of 3 had had heart attacks with pounding, heartache, fear and dyspnea every two or three weeks. These attacks were sometimes associated with left-sided frontal headache, nausea and vomiting. They lasted twenty-four to forty-eight hours, and were followed by complete recovery. A positive Chvostek sign with increase of all tendon reflexes was noted in this patient. A similar case with alternating heart and stomach attacks is reported. The author calls these attacks migraine with angina pectoris nervosa. He states that they are less frequent in adults and that one should consider cardio-neurotic attacks at this age, as equivalents of migraine. It is proper, according to the author, to consider migraine attacks, like asthma, hiccup, mucous colitis and other vasosecretory attacks, as expressions of an anaphylactic shock. Among other anaphylactogenic conditions associated with the attack, the author includes circumscribed edemas, and reports two such cases. In one case, in a girl of 12, a swelling of the left shoulder with fever had occurred periodically in association with headaches and nausea since the age of 1 year. In another case, periodic swellings occurred in the right upper arm with left-sided headache, amaurosis and nausea. Such attacks of edema, especially in the face, are not infrequent in adults. In children they are uncommon and may be confused with articular rheumatism, joint disease and the like.

In this same group the author places periodic skin eruptions such as blisters and herpes. A rather stereotyped case of this kind is reported: A child, four years of age, had had regular large blistering eruptions since the age of 1 year, at times associated with swelling of tonsils and cervical glands. It also suffered from unilateral headache with amaurosis and vomiting. Curschmann has seen only one such case in an adult, in whom the blisters on the left hand and foot were hemorrhagic in character. Perhaps such skin affections may also occur as genuine periodic equivalents of migraine.

The author emphasizes the fact that many of these cases of migraine show a positive Chvostek sign. The occurrence of migraine with frank spasmophilic epilepsy cannot be doubted. The author reports such a case of migraine with spasmophilic epilepsy in a girl of 13. Following calcium therapy the headache diminished and, in place of convulsions, slight depression with hesitancy of speech and fatigue occurred.

While the close relationship between epilepsy and migraine is known, it is not so clear that a relationship exists between spasmophilia and migraine. The frequency of the positive facial phenomena, and in some cases the presence of galvanic overexcitability direct attention to the endocrine components of childish migraine. The author deems it possible that a hypoparathyroid constitution with its associated vagatonia may be the basis for the existence of a hemicrania, just as bronchial asthma may result from an endocrine disharmony and anaphylactic reactions. It is hardly justifiable to consider migraine the product of hyperthyroidism, for migraine is not associated with myxedema or thyroidism to any degree.

The author advises the use of calcium in the migraine of childhood. It is the only medication from which he has seen specific results. What this means

for hemicrania may be measured by the meager results which are obtained from the therapy of migraine in adults.

Another rare yet interesting form of migraine in childhood is one accompanied by a slight vestibular irritation in conjunction with the attack. The author reports a case in a child of 14, who, since the age of 6, every two or three months has had a right-sided headache with nausea and horizontal vertigo. The neurologic examination revealed a definite horizontal nystagmus with increased Bárány responses. A sister, aged 17, has also had migraine since childhood. The mother had headaches and vomiting. The maternal grandmother had similar attacks, associated with vertigo. This case, according to the author, presents a classical example of an homologous inheritance of a distinct type of heredodegeneration or vestibular migraine. Vertigo is rather rare in children. That sporadic hemicranias with more or less marked Ménière syndrome occur in adults is certain. Also in organic affections, such as otosclerosis, migraine is not infrequently found, but no doubt in them it is a coincidence of two frankly constitutional degenerative manifestations. The author has also noted the relationship between vestibular phenomena and tetany attacks. From a therapeutic standpoint, the vestibular type of migraine in children may be treated with the vestibular sedative, quinin.

The author reports two cases of skull abnormalities or so-called "Turmschädel" in the migraine of childhood. The reason for reporting these two cases is that these observations appear to have corroborated the mechanical genesis of Sculler and Auerbach who attribute the cause of migraine to a lack of proportion between brain volume and skull circumference. Hemicranias without skull anomalies and vice versa are so frequent that the pathogenic importance of skull asymmetry is to be doubted.

In conclusion it is pointed out that no clear relationship exists between migraine in children and the acute infectious diseases. It is well to bear in mind the forms it may assume and that the history is often the only clue to the diagnosis.

MOERSCH, Rochester, Minn.

STUDIES IN EXPERIMENTAL TRAUMATIC SHOCK: VI. THE ACTION OF ETHER ON THE CIRCULATION IN TRAUMATIC SHOCK, McKEEN CATTELL, PH.D. Arch. Surg. 6:41 (Jan.) 1923.

It is a long-known and well-recognized fact that patients in shock are poor anesthetic risks. This study was undertaken in an attempt to determine the effects of ether on the factors which influence the blood pressure, with special reference to the conditions as they occur in shock or circulatory failure. The effect of ether on the blood pressure and on the integral parts that go to make up the blood pressure—the heart, vasomotor system, and blood vessels—and finally the circulation as a whole, with the changes that occur in shock, are considered.

From a consideration of the literature, Cattell believes that ether, if properly administered to normal subjects, produces no fall in blood pressure and may even cause a transitory increase during the early stages of its administration; whereas, in shock, circulatory collapse frequently occurs.

As it has been abundantly proved that a condition of light anesthesia can be brought about with practically no effect on the blood pressure of the normal animal—the object of these experiments was to study, not the effects of light anesthesia, but rather the acute effects of concentrations of ether having a definite influence on the blood pressure. Cats were used throughout as the

experimental animal. As the preliminary anesthetic, light ether, light ether followed by decerebration, or urethane was employed. The disappearance of the corneal reflex was used as the standard of the depth of the anesthesia.

In the normal animal, the sudden inhalation of strong ether results in a sudden drop in the arterial pressure, which is quite temporary. As the anesthesia deepens, the pressure gradually recovers until, by the time the eye reflex has disappeared, it may have returned to its original level. By giving the ether gradually, it is always possible, in the normal animal, to attain a depth of anesthesia in which the eye reflex is abolished, without the intervening drop in blood pressure. If ether is inhaled while the animal is anesthetized, the effect on the blood pressure is very much less than when it is given to an animal that is not already under the influence of ether. Similarly, the fall in pressure caused by the intravenous injection of ether is much less in the deeply anesthetized animal. But in the shocked animal, regardless of the speed of the administration of the ether, the blood pressure will show a considerable decrease at the point when the eye reflex is just obliterated. Nitrous oxide-oxygen anesthesia, in the proportion of three to one, will produce a full degree of anesthesia without any fall in blood pressure.

In an attempt to discover a cause for this fall in blood pressure, observations were carried out on the heart volume of intact cats by an ingenious method whereby, by means of the insertion of a cannula through the pericardium, the pericardial sac is made to function as an oncometer and register volume changes in the size of the heart. From these observations on the heart volume of intact cats and from a consideration of the blood pressure readings made at the same time, it is apparent that the administration of ether results in a depression of the heart and a decrease in its output, which is sufficient to account for the fall in pressure in both the normal and shocked animals. In confirmation of this observation, the effect of ether on the isolated cold blooded heart was noted. It was found that when immersed in salt solution containing 0.18 per cent. ether by weight (a percentage that corresponds to the concentration of the ether in the blood of the moderately anesthetized animal), a marked reduction in output with a decrease in the extent of the contractions is produced.

An injection of epinephrin will apparently reduce the sensitivity of the heart to ether. The effects of an overdose of ether on the heart are much less marked for an hour or more following the administration of this drug. It is suggested that this is due to the antagonistic action of ether on the heart. Pituitary extract does not influence the pressure drop produced by ether in the shocked animal.

To determine the effect of ether on the tone of the arterioles, observations were made on the leg volume as indicated by the plethysmograph. The results showed that the administration of ether, as in ordinary anesthesia, results in a constriction of the peripheral vessels, which is mediated through the vasomotor nerves. Perfusion rate experiments, and results obtained by the injection of ether directly into the circulation, together with the form of the blood pressure curves, indicate that ether causes a contraction of the peripheral vessels. This constriction may be due to either a direct stimulation of the vasomotor center, or a reflex to the fall in pressure resulting from a depression of the heart. In the normal animal, therefore, the primary drop in the blood pressure curve following the administration of ether is probably due to an influence on the heart; and the secondary recovery is due to a compensatory vasoconstriction.

Any circumstance which tends to depress the general condition of the animal such as low blood pressure, hemorrhage, severe operations, or the injection of acid into the circulation, seems to produce the condition of ether sensitiveness. In the shocked animal, no evidence of vasoconstriction produced by ether was obtained, and pressor effects from asphyxia or sensory nerve stimulation became less or were entirely absent. Therefore, the cause of the greater depressing influence of ether on the blood pressure in shock seems to be a disturbance of the vasomotor system. The usual compensatory constriction no longer occurs to offset the decreased output of the heart, and the pressure continues to fall. This might be due to a depression of the vasomotor center or to an already existing maximum tone of the center so that there could be no compensation.

GRANT, Philadelphia.

TUMOR OF THE INFUNDIBULUM (TUBER CINEREUM), INTEGRITY OF THE HYPOPHYSIS, ADIPOSE SYNDROME. RODOLPHE LEY, *Rev. neurol.* 29:376 (April) 1922.

This is a case report of a girl aged eleven in whom necropsy revealed a tumor of the infundibulum, with an intact hypophysis. The interesting point in addition is that there was a typical adipose syndrome during life. The family history was negative for nervous and mental disease, except for a sister who had some cerebral disorder described as "meningitis." The first noteworthy symptom in the case was a strong tremor involving the upper extremities, appearing at about the ninth year. This tremor prevented attempts at writing as the child grew older. After this, tremor difficulties in gait appeared, and still later speech disorders. In her eleventh year, three months before entering the hospital, she vomited almost daily. Before this, intellectual defects were quite marked. She also had been given to bed-wetting, and there was a recent history of two or three hallucinatory episodes or crises.

Objective examination showed no stigmata of degeneracy, nor of congenital syphilis. There were no paralyses, nor muscular atrophies. There were tremors of all parts of the body except the head, which were increased by an effort at voluntary movement. No Romberg sign was present. All tendon reflexes were abolished. The external ocular muscles were normal; pupils, large and equal, but reacted feebly to light and accommodation. Speech was scanning in type. There was no sensory disturbance. A Babinski sign was impossible to demonstrate because the toes were in continuous extension. Vision was defective, but the ocular fundi were negative on ophthalmoscopic examination. The cerebrospinal fluid was negative, except for a positive Ravaut test. Toward the end stage all symptoms were aggravated: vomiting was frequent, walking was impossible, and the patient was emotionally indifferent; finally there developed loss of sphincter control and decubitus.

Clinically, multiple sclerosis was ruled out because of the absence of nystagmus, and contractures or spasticity. The case looked like a cerebral tumor despite the normal appearance of the fundi. Localization was difficult. The prerolandic area could not be considered in the absence of a condition of equal intensity on both sides. Involvement of the frontal region, and, because of the tremors, of the basal ganglia had to be considered. Hence the necessity for depending on the necropsy for the final diagnosis, especially as to localization. This showed a tumor of the infundibulum with an intact pituitary body. On sectioning, definite sclerosis of the right temporal lobe and right thalamus

was demonstrated macroscopically. Moreover, there was this peculiar condition: The white matter had a grayish appearance, and the gray substance had the appearance of white substance.

Microscopic examination revealed in the main: Changes in all endocrine glands except the ovaries and hypophysis; profound neuroglial proliferation in the nervous tissue, particularly in the cerebrum; marked gliosis of the tuber cinereum; neuroglial hypertrophy of the internal capsule; gliosis of the lenticular and caudate nuclei, with frequent giant neuroglial cells; rarified cells in the dentate nucleus; macroscopic abnormality of the locus niger; it was grayish instead of black, and microscopically showed a diminution in the number of cells, which were for the most part void of pigment.

In a general way, there was diffuse gliomatous change of almost all areas of the forebrain and midbrain, with added features of a tumor of the infundibulum, in a child with marked obesity. To this is to be added the fact (in harmony with experiments in animals) that a tumor of the cinereum is incompatible with life. Further, it is to be noted that a lesion of the infundibulum caused hypophyseal symptoms, with an intact hypophysis. This means that the teachings on the physiology of the hypophysis must be revised

JONES, Detroit.

A CORRELATIVE STUDY OF ENDOCRINE IMBALANCE AND MENTAL DISEASE. NOLAN D. C. LEWIS and GERTRUDE R. DAVIES, *J. Nerv. & Ment. Dis.* **54**:384 (Nov. and Dec.) 1921, and **55**: (Jan.) 1922.

The introduction takes up briefly the history of endocrinology and its supposed relation to behavior. The investigation was for the purpose of determining the relationship between the physical manifestations, blood chemistry and mental syndromes in hypotonic and hypertonic groups and was based on the following characteristics: abnormal growth conditions of the skeleton, muscular dystrophies, skin condition, trophic changes in hair and nails, altered action of sweat glands, sluggish circulation, subnormal pulse, temperature and blood pressure, early arteriosclerosis, dysgenitalism, obesity, nocturnal enuresis, polyuria or glycosuria, fatigue without energy expenditure and enlargement of the thyroid with associated phenomena. The following tests were performed on each individual to aid in classification as hypo-endocrinic or hyperendocrinic function: Sugar tolerance, thyroid function test (the technic of which is given), blood examination for uric acid, urea nitrogen and creatinin.

Case histories then follow, the mental and endocrine diagnoses of which were as follows: Schizophrenia with projection and hyperthyroidism; schizophrenia with projection and polyglandular syndrome; schizophrenia and periodic hypoadrenia; schizophrenia with projection and hypothyroidism with gonadal hypofunction; schizophrenia with projection and submyxedema with hypoadrenia; schizophrenia with projection and hypothyroidism with obesity; schizophrenia with inversion and hypothyroidism; affective schizophrenic psychosis and hyperthyroidism; paranoid schizophrenia and mixed thyrotrophic type; schizophrenia with projection and hypothyreosis with hypoadrenia; schizophrenia and hypothyroidism with hypoadrenia; defective schizophrenic epilepsy and dyspituitarism; affective schizophrenia and periodic hypoadrenia; schizophrenia and hypothyroidism; paranoid schizophrenia and hyperthyroidism; schizophrenia with projection and hyperthyroidism; schizophrenia with projection, hypopituitarism and hypothyroid-

ism; schizophrenia and hyperthyroidism associated with pituitary dysfunction; schizophrenia with projection and hypothyroidism; schizophrenia with catatonia and hypothyroidism; and schizophrenia with introversion and hypothyroidism. At the end of each presentation is given an outline considering behavior, physical findings and laboratory results together with results of endocrine glandular therapy, surgery or other treatment that was given.

A table and charts are given under the heading of discussion. Urea was somewhat higher than normal in all cases. Uric acid and creatinin were about normal, but sugar tolerance and the thyroid test possibly gave some diagnostic aid in determining the type of endocrine response. As to sugar tolerance, there was delayed absorption in the four hyperthyroid cases. The only other similar reaction was in a pluriglandular case. In each of the twelve cases of hypothyroidism sugar tolerance was increased. A similar reaction was found in two cases of hypoadrenia, one hypopituitary case and one polyglandular case.

Three of the four hyperthyroid cases gave a hyperglandular type of reaction to the thyroid tests. One was not tested (exophthalmic goiter). Eleven of the hypothyroid cases gave the hypoglandular reaction (no change in pulse rate—the normal cases showed an increase). The other hypothyroid case did not cooperate. The remaining six cases (hypoglandular and mixed types) gave a hypoglandular reaction. In one there was a contra-indication.

The authors state "It has been discovered that, regardless of the type of hypofunction, the preliminary administration of thyroid gland over a period enhances the action of the other glands to be given later." Contra-indications are exophthalmic goiter, manic or anxiety conditions and advanced valvular heart disease.

Of the seventeen thyroid cases reported, eight, with psychoses of from nine to two and a half years, were benefited physically and mentally; seven were improved physically but showed changes in behavior not recorded as improvement. The other two did not receive treatment.

A table is given showing the results in the twenty-two cases; eight were improved mentally and physically and seven were improved physically but not mentally, two showed no change either physically or mentally, one improved mentally but not physically and four were discharged as recovered.

In conclusion the authors state that: (1) due to the close association of the activities of the nervous system, particularly the autonomic system with endocrine function, we would expect a response in the glands to maladjustments of the individual to certain situations, and that glandular defects would produce peculiarities of behavior and limitation in the action systems. In mental disease there is an abnormal circle established, the areas of which are composed of both factors; (2) the effects of glandular disturbance are due to a change in the rate of normal function; (3) for every case of profound glandular disturbance there are dozens with very few signs, in which psychic or chemical treatment is most likely to be successful; (4) the sugar tolerance test and the thyroid function test are extremely valuable in diagnosis and in the cases in which the fewer signs are found, there is the earliest and best opportunity for glandular therapy; (5) occupational therapy was useful from a therapeutic standpoint and also as an early indicator of variations in behavior.

CHAMBERS, Philadelphia.

NEUROPSYCHIATRIC SEQUELAE OF ACUTE EPIDEMIC ENCEPHALITIS IN CHILDREN. FRANKLIN G. EBAUGH, Am. J. Dis. Child. 25:89 (Feb.) 1923.

The psychiatric and neurologic sequelae of a group of seventeen children referred from school, home and hospital to the Neuropsychiatric Clinic of the Philadelphia General Hospital are considered. There were six girls and eleven boys from two to fourteen years of age and the lapse of time from the subsidence of the acute encephalitic symptoms was: 4 cases, three or more years; 2 cases, two or more years; 5 cases, one or two years; and 17 cases, less than one year.

*Psychiatric Sequelae.*—(1) A total change in character and disposition was present in ten cases, and was evidenced by hyperkinesis, emotional lability, irritability, affability and over-affectionateness; sexual precocity was also noted. Three patients showed no behavior abnormalities after the termination of the acute disease. Nearly all cases were corrigible. (2) Nocturnal restlessness and insomnia is followed by sleep into the forenoon. This persistent insomnia rarely occurs in children with other diseases; it is sometimes seen in severe chorea. According to the author, it is a manifestation of a neurotic condition; he says: "Insomnia is a common sequela of acute encephalitis in children." (3) Affective disorders are an important part of the change of disposition already referred to. Three patients attempted suicide, but there was no real desire to commit suicide comparable to that in depressive reactions and the characteristic slowness of thinking and planning peculiar to depression was absent. (4) Hysterical reactions were present in three cases: one had prolonged periods of rapid respiration; another, spells of screaming and yelling; the third had shown hysterical tendencies—spells of blindness, deafness and aphonia—before the encephalitis, but all were aggravated by the attack. These reactions were efforts to avoid school, discipline or unpleasant duties. (5) Tics of the head, of one extremity, sniffing of the nose, clicking of the tongue, biting the nails, and spitting were observed; these were of organic origin. (6) Mental deficiency was present in three cases: one case four years after encephalitis showed a mental age of two years, the age at which the disease had begun; another, in which the encephalitis occurred at birth, two years later was an idiot. This case suggests that encephalitis may be congenital.

*Neurologic Sequelae.*—The subjective sequelae were: headaches, dizziness, diplopia, diurnal drowsiness, asthenia, and sialorrhea. The objective sequelae were: (1) Disturbance of motility—choreiform and other types of incoordinate movements—occurred for prolonged periods in seven cases. The movements were brought on by emotional stimuli, and occurred frequently during sleep. Pain was rare. Radiculitis was present in one case only in which choreo-athetoid movements were also found. Tremors were frequent but not characteristic; (2) Visual disturbances: seven cases showed weakness or paralysis of one, most frequently the external, rectus muscle; ptosis was present, though not in marked degree, and was permanent in two cases; eye ground examinations were practically negative. (3) Other neurologic disturbances: facial weakness in two cases; pyramidal and extrapyramidal tract involvement in one case, in which there was rigidity of one side of the body and immobile facies; this was the only case which showed a paralysis agitans syndrome. Sialorrhea, vasomotor symptoms and exophthalmos occurred in one case; hypopituitarism was present in one case; and obesity developed in several cases.

*Prognosis.*—Ebaugh states that the prognosis "should be made very conservatively," though he wishes to avoid a "pessimistic outlook." Five cases have been progressively improving, four are improving slowly, and eight are unimproved, three of the last being permanently, mentally deficient. A period of five years, he says, "should be allowed to determine the permanency of the neuropsychiatric sequelae."

*Treatment.*—There is no specific remedy; medicine is of no avail. Sedatives and hypnotics have not produced sleep, though warm baths have been of use; spinal drainage has given conflicting results. Psychotherapy has been disappointing, but occupational therapy has been valuable. Prolonged observation and hospital treatment are advisable and this is one aspect of the need for a childrens' psychopathic ward. Commitment is sometimes indicated. Well regulated home routine and discipline were advised especially for cases with hysterical tendencies.

CHAMBERS, Philadelphia.

WAR AND ACUTE DELIRIOUS HYPERTHYMIA. R. BENON, Rev. neurol. 28:1011 (Sept.-Oct.) 1921.

The first paragraph in the form of a "summary" gives the gist of the paper. It refers to: the historical consideration of the subject; emotional shock, or repeated emotions, as one of the results of battle; obsessing melancholy, or depressive memories, etc. It then enumerates acute delirious hyperthymic manifestations, including delusions, hallucinations, and illusions. The paper records two cases with observations. The evolution of the mental state is varied. The article gives a criticism of the emotional syndrome of such cases, and affirms that the true psychoses are not peculiar to war.

The two cases recorded are quite different in some of their chief symptoms, though they present many features in common: Both patriots had been through some of the worst horrors of the World War; both were so severely depressed as to be incapacitated by their emotional reactions and to require hospitalization; both passed through a delirium, with the accompanying delusions, hallucinations, illusions, etc. But, the first case, a young sergeant, 26 years of age, recovered sufficiently to be sent back to the front where he died in action, and was after his death cited for unusual bravery. His family history and personal make-up showed no history of taint or defect. The second, a private, 40 years of age, a carpenter by trade, gave a history of hereditary taint, one sister having been committed to a hospital for the insane, and one brother having shown distinct hypochondriac tendencies. The patient himself had contracted a chancre at 26, and his wife later had two miscarriages. The later history of this patient is quite different from that of the first. While he showed similar nervous and mental symptoms in the earlier period of his illness, he never made a complete recovery; his emotional troubles, however, chiefly anxiety symptoms, had disappeared by 1916. (Both patients had entered the service in 1914). His general asthenia with irritability tended to pass into a chronic state. As late as March, 1920, he showed a "post-emotional chronic asthenia." There were marked character changes such as impatience, easy irritability, and tendency to isolate himself from others. He had frequent headaches and discomfort in the spine, and he tired easily on effort. He became sexually impotent and worked little or not at all until his wife finally had to seek medical aid.

After several careful readings of the paper, it must frankly be confessed that there is little in the cases that we do not frequently see in the clinics of our large hospitals. Moreover, it is not clear in the second case that some

of the clinical symptoms and complaints may not have been due to the syphilis contracted fourteen years before the patient entered the service, although it must be admitted that the general condition may have been precipitated or aggravated by the experiences of war.

The author brings out the important point that on the primitive hyperthymic state there are grafted illusions, hallucinations, delirious interpretations and the like, especially in the first case, even including ideas of persecution. He also admits that there is a predisposition to such symptoms in the second case, though he does not ascribe it in any way to syphilis, but rather implies that heredity plays an important part.

He also lays emphasis on "emotional syndrome" in both cases, but concludes that this is not peculiar to war; any sort of catastrophe might precipitate a similar syndrome. In his last sentence he states that "acute delirious hyperthymia" due to war differs in no way, except in "color and form," from that seen daily in other delirious states. In his two cases he thinks the emotional states are peculiar in that their particular characteristics are due to individual predispositions—congenital or acquired.

JONES, Detroit.

WASSERMANN REACTION AND COLLOIDAL METHODS IN THE SPINAL FLUID OF NEUROSYPHILITICS. THEORETIC CONSIDERATIONS ON THE WASSERMANN TEST. C. Rizzo, Cervello, 1:5 (Sept.-Oct.) 1922.

This paper contains a thorough review of work on this subject in different countries and a report of the personal experience of the author. Some clinical cases are reported. Ample, accurate bibliographic data close the article. The conclusions are: The colloidal reactions (gold, mastic, Prussian blue, benzoin etc.) on spinal fluid, are not specifically indicative of cerebrospinal syphilitic lesions. They do not differentiate with certainty the various forms of syphilis of the nervous system. They are indications only of an inflammatory or irritative state of the central nervous system. The positive Wassermann reaction in the spinal fluid is the only sure index of a neurosyphilitic lesion. Minimal traces of blood mixed in the spinal fluid disturb the results of the colloidal reactions, but they have no influence whatever on the Wassermann reaction.

The result of the Wassermann test on the spinal fluid has always a greater value than the result of the blood test. The latter may lead to erroneous interpretation. A negative blood Wassermann reaction cannot exclude a syphilitic infection; a negative Wassermann reaction with the spinal fluid excludes the existence of serious active syphilitic processes in the central nervous system.

There are spinal fluids of neurosyphilitics with positive Wassermann reaction, which become negative after inactivation at 56 C. for half an hour (phenomenon of thermolability); it is therefore necessary, especially if only a small quantity of spinal fluid is available, to try first the reaction on the active fluid; and, if the quantity is sufficient, in a second examination to repeat the reaction on the inactivated fluid. In giving the report of a test, it is always necessary to indicate whether the blood serum or the spinal fluid was active or inactivated.

Spinal fluids from neurosyphilitics which give a positive Wassermann reaction when active, and a negative reaction when inactivated, give, in both states, a positive colloidal benzoin reaction; in other words the results of the two reactions are not parallel.

When the Wassermann reaction, positive in the active spinal fluid of a neurosyphilitic with high cell count, becomes negative after centrifugation and inactivation of the fluid, it becomes negative also in the same inactivated fluid without centrifugation; that is, the transformation of the reaction from positive to negative after inactivation takes place either in the presence or the absence of lymphocytes in the fluid, with or without their autolysis.

The amount of albumin in the spinal fluid has no influence whatever on the Wassermann reaction. The xanthochromic hyperglobulinic fluids, which sometimes have an albumin content of 36 gm. per liter and more, generally present a negative Wassermann reaction; in the rare cases in which it has been found positive, it became negative after inactivation of the fluid; for this reason such a reaction has not been considered specific. If the Wassermann reaction, found positive in spinal fluids that are neither xanthochromic, nor hyperalbuminous, nor coagulable, becomes negative after inactivation, it is specific, and ordinarily is accompanied by other alterations of the fluid (lymphocytosis, increase of globulins, etc.).

The two varieties of syphilitic reagents (thermolabile and thermostabile), which, according to some investigators, are found separately or in various mixtures in the blood and in the spinal fluid, do not exist as biologic entities. The phenomenon of thermolability may be found in the same individual at the same time in the blood serum and spinal fluid, in the spinal fluid alone, or in the blood serum alone.

The general paralytic spinal fluid does not present the phenomenon of thermolability; the intensity of the Wassermann reaction in the inactivated fluid of a general paralytic is the same or slightly less than that of the active fluid.

In cases of cerebral syphilis and uncomplicated tabes, the author has been able to find the phenomenon of thermolability in the spinal fluid; he considers this a differential diagnostic symptom and a good prognostic sign.

NACCARATI, New York

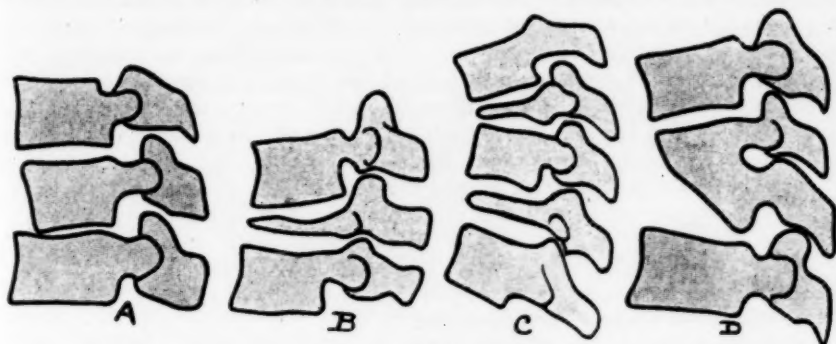
THE RADIOLOGIC SIGNS OF VERTEBRAL CANCER. SICARD, LERMOYEZ and LAPLANE, *Ann. de méd.* 13:383 (April) 1923.

This is a concise exposition of the differences between the radiologic signs in tuberculous spine and in vertebral cancer. The first radiologic sign in Pott's disease is a thinning of the intervertebral disk, which gives the impression of being pinched between the two adjacent vertebrae. This is especially visible in the lateral view and precedes by a considerable time any changes in the transparency of the vertebral bodies. The early thinning and deformation of the cartilage is shown in schematic outline at *A* in the accompanying illustration. In contrast, in vertebral cancer, the rule is complete integrity of the intervertebral disks.

In vertebral cancer one finds in a vertebral column of which the vertebrae are normal and the disks intact, one vertebra smaller than the others, very notably diminished in height, especially in the anterior portion. Such a vertebra marking the focus of cancerous destruction is apt to appear as a cuneiform mass with the summit directed anteriorly. When the bony destruction is considerable, the affected vertebra appears as a thin plate of which the height is hardly half of that of a normal vertebra. This is schematized in *B* of the illustration.

In *C* is outlined the deforming process when two metastatic cancerous processes have involved two vertebrae, which are not adjacent, simultaneously.

In Pott's disease, there can occur a false appearance of intact disks as shown in *D* of the accompanying illustration. In reality, the mass in the form



Outline of roentgenograms: *A*, Pott's disease; *B*, vertebral cancer; *C*, cancerous disease of two vertebrae; *D*, Pott's disease with consolidation of two vertebrae.

of a wedge is formed of two vertebrae almost completely consolidated; this is because one intervertebral disk has been very seriously changed and partly destroyed.

DAVIS, New York.

EXPERIMENTS ON THE TRANSPLANTATION OF THE SPINAL CORD IN AMBLYSTOMA, AND THEIR BEARING UPON THE STIMULI INVOLVED IN THE DIFFERENTIATION OF NERVE CELLS. S. R. DETWILER, *J. Exper. Zool.* **37**:339 (May) 1923.

Hyperplasia of both sensory and motor nerve cells in *Amblystoma* can be experimentally induced, but the stimuli involved in each case are not the same. Hyperplasia of the sensory nerve centers takes place in response to increased functional demands at the periphery, but the added demands of a terminally increased musculature do not produce a similar response in the motor nerve centers. Extirpation of the anterior limb rudiment in *Amblystoma* before the outgrowth of the peripheral nerves is followed by a hypoplastic development of the peripheral afferent neurons of the brachial level of the cord. Motor cellular differentiation within the limb level of the cord appears to be unaffected by the extirpation of the limb, as evidenced by a numerical comparison of the cells in the two halves of the cord at the level involved. When the anterior limb is transplanted caudad to its normal site on the same embryo, the peripheral nerves supplying it are found to be larger than their counterparts which have no connection with a limb. This enlargement is found to be the result of an increased number of peripheral afferent neurons which have developed in response to the additional demands of the increased sensory area. The peripheral efferent neurons, however, fail to undergo a similar hyperplasia under the increased functional demands of the added musculature of the transplanted limb and shoulder.

Motor hyperplasia has been produced by excising the limb level of the spinal cord (third, fourth, and fifth segments) and transplanting into the excavated

area, a more caudal unit of the cord (seventh, eighth and ninth segments) taken from another embryo. The neuroblasts in the grafted unit of cord are found to undergo increased development approximately equal in extent to that which characterizes the normal brachial enlargement. The results of the spinal cord transplantation experiments indicate that motor cellular differentiation within the spinal cord is determined primarily by functional connection with the central longitudinal reflex pathways. This is suggested in the fact that the seventh, eighth, and ninth segments, which fail to undergo motor hyperplasia when in connection with a transplanted limb, will undergo marked hyperplasia when subjected to the stimuli normally activating the increased production of efferent neurons at the limb level. The mechanism involved in the production of increased motor cellular differentiation at the limb level, as evidenced by these experiments, appear to fall in line with the stimulo-genous fibrillation concept of Bok (1915), in that the greater stimulation passing through a specific central conduction pathway, which normally terminates at the limb level, activated the outgrowth of peripheral motor axons in a ratio proportionate to the number of fibers normally present.

The normal character of the brachial plexus developed from the grafted unit of cord (seventh, eighth, and ninth segments), indicates that the final architectural plan of the plexus and its derivatives is determined by the mechanical factors involved in the differentiation and growth of the structures with the appendage. There is no evidence to indicate that they influence, in any way, initial outgrowth of the nerve fiber, nor the establishment of primary connections at the periphery. The behavior of the early embryos with composite spinal cords, in response to tactile stimulation, bears out the architectural scheme of the primary reflex mechanism as outlined by Herrick and Coghill (1915). The experimental observations above recorded suggest that the caudal development of the ventral commissure follows very quickly after its functional establishment at the anterior end of the cord.

WYMAN, Boston.

#### FAMILIAL MYOPATHY OF THE PERONEAL OR DISTAL TYPE.

LOUIS RIMBAUD and GASTON GIRAUD, *Rev. neurol.* **28**:1004 (Sept.-Oct.) 1921.

The opening paragraph gives a general summary of the disorder mentioned as illustrated by three cases, two of which are strikingly similar. The syndrome of progressive amyotrophy of the adolescent period occurring in three brothers, with negative heredity, showed itself from the first as a distal or peripheral disorder in the lower extremities, and then later in the upper extremities, with a predominance of the atrophy in the extensors. There was intense and marked muscular contraction and increase of fibrotendinous tissue especially in the flexors, and a complete absence of neuritic or myelitic symptoms: i. e., no fibrillary contractions; no neuromuscular hyperexcitability of any kind; no qualitative alterations of electrical reactions; no tendon reflex disturbances, sensory disorders, sphincter disturbances, or abnormal pyramidal tract signs. In short, the condition described is a progressive myopathic amyotrophy with a tendency to an increase of fibrous tissue, revealing by its peripheral or distal distribution the topographical appearance of the peroneal myeloneuropathic syndrome of Charcot-Marie-Tooth. In other words, there is an apparent discrepancy between the topographical distribution of the myopathy and the intrinsic neurologic characteristics.

There follows a detailed examination of the three cases including movements, attitude, trophic muscular state, electrical reactions, gait, sensory condi-

tion, trophic condition of the skin, tendon reflexes, idiomuscular excitability, cutaneous reflexes, etc., with the general conclusion noted above. One of the most notable features is that the change in motility is proportional to the degree of muscular atrophy and appears to be conditioned by it.

Two different types of muscular atrophy are then discussed a little more in detail from the theoretic point of view: the Charcot-Marie-Tooth type; and the myopathy of the peripheral or distal type. Only nine cases of the latter have been reported, beginning with Oppenheim and Cassirer (1898) and ending with Spiller (1913), and Cockayne (1913). The essential point brought out is that a true myopathy is essentially a disease of muscle tissue. Then are given the differing opinions of various schools, such as that of Charcot-Marie, on the one hand, and Dejerine, Hoffman, and others, on the other. The writers lean to Grasset's opinion that it is very difficult to discriminate between a central lesion and disorder of the axon from central cells. He sees in the neuron a physiologic and anatomic unit, and considers the peripheral or distal type of amyotrophy as secondary to a chronic disorder, which in turn is quite different from a primary or essential amyotrophy. It is only by slight or almost insignificant neuritic signs that one can make the differential diagnosis, independent of the topography. The absence of such signs in the three cases under consideration is notable. (1) Fibrillary contractions are wanting; (2) there is no idiomuscular hyperexcitability; (3) the electrical reactions show only qualitative alterations; (4) the tendon reflexes are preserved even in an advanced stage of the atrophy; (5) pyramidal tract reflex disorders such as the Babinski sign are not present; (6) the fibromuscular and tendon distortions are quite striking. (The paper is illustrated with photographic reproductions of these changes.)

JONES, Detroit.

MENTAL DISORDERS OF CHILDREN. BERNARD GLUECK, State Hosp. Quart. 8: No. 2, (Feb.) 1923.

"A clear cut and relatively well organized statement of psychopathological issues such as is possible to a very large extent in connection with adults is still an unrealized ideal in the domain of childhood." This condition has the advantage of allowing one to follow uncritically the mental disorders of childhood, but makes the task of child psychopathology difficult. The author clearly emphasizes the necessity of viewing the child as an integrated unit and of studying the problem of the "personality as a whole." The underlying meaning of personality difficulties in childhood must be searched for in the child and the rôle these play in efforts for adaptation to the environmental demands must be considered.

One commonly meets with a multiplicity of causes for the psychopathologic disorders of childhood. Heredity and constitutional elements may play a part but the events and experiences in the life of the individual "may be found to have been operative in causing the condition." The latter conditioning influences guide one's management of childhood problems and are more productive than theories concerning heredity and predestination. Much of the psychopathology of childhood may be explained in terms of unhealthy conditioning influences of life and "unhygienic habits of biological and social adaptation."

On the basis of our present knowledge no rigid scheme for use in dealing with childhood psychopathology can be set forth. The author suggests the

following grouping: (1) arrest of intellectual development of various degrees of intensity: (a) idiocy, (b) imbecility, (c) moronism, (d) dull normality; (2) the constitutionally psychopathic children; (3) the psychoses of childhood: toxic-exhaustive deliria, manic-depressive states, schizophrenic states, syphilitic disorders; (4) organic nervous disorders of childhood; (5) functional nervous disorders of childhood: (a) psychoneurotic reactions, (b) reactive manifestations: convulsive phenomena, spasms and tics; speech disturbances; sleep disturbances; kinetic disturbances; (6) conduct disorders of childhood; (7) endocrinopathies of childhood.

EBAUGH, Philadelphia.

ZONA. RAMOND, *Progrès méd.* **38**:97 (March) 1923.

This is an interesting clinical discussion from which a few points only will be abstracted. The forme fruste of zona—herpes zoster without skin eruption—is a clinical certainty, as shown by one case reported by the author. In this man, the pain was sharply localized to four intercostal spaces. There was no eruption at any stage—the case was followed through to cure. But at the level of the neuralgic pain there was a band of hyperesthesia both to tactile and thermal stimuli, the pupil was dilated on the affected side (sign of Griffo), and there was a low lymphocytosis in the spinal fluid. These points justified the diagnosis.

The author considers that spinal fluid lymphocytosis is frequent, but not constant, in herpes zoster and that its occurrence depends on whether the internal or the external portion of the posterior ganglion is affected. The internal portion is intra-arachnoid; affection of it is therefore accompanied by spinal fluid lymphocytosis. The external portion of the ganglion is extra-arachnoid and concomitant cellular changes in the fluid are not present.

The author considers that each portion of the posterior ganglion corresponds with a precise cutaneous territory. The internal portion has to do with the anterior part of the body surface. Therefore zona, with the skin eruption limited to the anterior part of the chest or body areas, is indicative of inflammatory processes of the internal pole of the posterior ganglion and there is an accompanying lymphocytosis in the spinal fluid. In contrast, in zona with posterior skin signs, one must predicate change in the external pole of the ganglion and expect no increase of cells in the fluid.

In a later portion of the paper the syndrome of the geniculate ganglion, (Ramsay Hunt) is considered.

DAVIS, New York.

ACUTE HEMORRHAGIC ENCEPHALITIS. JOHN A. TOOMEY, LEON H. DEMBO and GUTHRIE McCONNELL, *Am. J. Dis. Child.* **25**:98 (Feb.) 1923.

The authors report a case of acute hemorrhagic encephalitis in detail because of the comparative infrequency of this as a sequel of scarlet fever. The patient, aged 6, had been in good health until twenty-four days before admission. He then had an attack of scarlet fever which confined him to bed for three days, but seemed to be well after that. Three days before admission he complained of sore eyes, drowsiness and stiffness of the right leg. He improved with medication. On the first day he vomited, was feverish and developed a squint. Rigidity was present in the right leg. The following day he vomited more and was more lethargic, but could be aroused. That

evening he had convulsions which started with twitchings of the fingers and then became generalized. The convulsions persisted and there was incontinence of urine and feces. The patient lapsed into coma and died forty-five minutes after admission. The spinal fluid was bloody, this being ascribed to faulty technic in withdrawal.

The necropsy findings were: The ventricles of the brain were filled with blood and blood clots; the basal nuclei were soft, friable and hemorrhagic, the right thalamus being more involved than the left; subependymal hemorrhages were present. The diagnosis was edema of the brain; acute hemorrhagic softening with intraventricular hemorrhages.

The authors discuss two distinct types of pathology: (1) that of Strümpell—Leichtenstein, the acute hemorrhagic type; (2) the type characterized by multiple hemorrhages.

The symptomatology in brief is usually that of an acute onset some time after recovery from an infectious disease, with headache, fever, restlessness, convulsions, rigidity and coma. Prodromal symptoms may be present. Ptosis, strabismus, pupillary inequality and loss of light reflex are common. Respiration is embarrassed. Terminal signs are increasing coma and respiratory failure.

CHAMBERS, Philadelphia.

#### THE RÔLE OF THE LABYRINTH IN EXOPHTHALMIC GOITER.

DENIS HELLIN and ADOLPHE SZWARE, *Rev. neurol.* **28**:1000 (Sept.-Oct.) 1921.

In a three page article the authors review the close association existing between disorders of the labyrinth and exophthalmic goiter. First, nystagmus is shown in from 34 to 60 per cent. if Stocker's test is used. In the second place, certain symptoms common to labyrinthine disorders and exophthalmic goiter are: hypotonicity and hypertonicity of muscles, asynergia, tremors, movements of the head, vertigo, vomiting, peristaltic disorders, acceleration of the pulse, etc., including the so-called sign of Moebius, and the labyrinthine sign of Bielschowski-Zacharczenko (convergence of the eye balls when the subject looks in a lateral direction). In the third place are the auditory symptoms: diminished hearing, tinnitus, combinations of suppurative otitis media and Ménière's disease. Exophthalmic goiter following explosions also indicates involvement of the auditory apparatus. Torticollis may occur in both disorders. Closely associated with this latter symptom in both disorders is strabismus.

Formerly the site of the nervous lesion in exophthalmic goiter was localized in the cerebellum, because of such symptoms as Babinski's adiadokokinesis. Experiments of Hogyes and Marikowsky have shown, however, that supination and pronation are dependent on the labyrinth. Likewise migraine was once traced to a cerebellar lesion, then later to Deiter's nucleus. From this it is only a step to the labyrinth. Kobrak's recent work has shown that migraine might well be due to disturbances of the circulation of blood in the labyrinth. Then too there is a close association between migraine and Ménière's disease. Finally, there is evidence that exophthalmic goiter is due to disturbance of the endocrine system, notably disorders of the suprarenals, and from this it is only a step to disorders of the labyrinth associated with disorders of the suprarenals. It is likely that certain toxins produce circulatory disturbances

in the labyrinth as shown by Curschmann and Kobrak. For example, nystagmus is produced by the action of the spirochete of Schaudinn, chloroform intoxication, alcohol, cocain, arsenic, etc.

JONES, Detroit.

A CONTRIBUTION TO THE STUDY OF APRAXIA. L. PAPADATO, *Encéphale* 18:253 (April) 1923.

This is a report of a case of brain tumor involving the left supramarginal gyrus and occupying the parieto-occipital area. The patient presented an interesting type of aphasia. There were: decrease in vocabulary with relative retention of spontaneous speech; comprehension of spoken language; alexia; and agraphia, although the patient was able to copy words and letters without understanding the meaning of the symbols.

In addition to this aphasia there was apraxia. Simple movements were performed equally well on both sides. Movements concomitant with emotional states were executed poorly on the left side and not at all on the right. On attempting to carry out complex acts there was perseveration and also inversion of the steps involved in a complex act. Imitation of movements was well done.

This apraxia, the author believes, is due to disturbance in kinesthetic sensation and might be called a sensory apraxia. It is quite distinct from true motor apraxia, which is due to loss of motor function, and is associated with a lesion near Broca's center. In addition to these "focal" forms of apraxia, the author admits the existence of a form associated with general intellectual deterioration, due to an extensive lesion interrupting long association tracts. The author states that when a lesion is confined to the association tracts of one hemisphere the apraxia will be more marked on the opposite side of the body. But if the commissural fibers in the corpus callosum have been involved, then the apraxia will be bilateral.

HYSLOP, New York.

THE SIGNIFICANCE OF THE TERM HIPPOCAMPUS. FREDERIC T. LEWIS, *J. Comp. Neurol.* 35:213, 1923.

The first definite description of the hippocampus of the human brain appeared in 1587 among the writings of Julius Caesar Arantius, a pupil of Vesalius. He compared this organ both to the sea-horse, *Hippocampus*, and the silkworm, *Vermis bombycinus*. A figure is shown of three images which Arantius may have had in mind: the mythical horse of Neptune, the sea-horse or true hippocampus, and two dolphins which were occasionally associated in mythology with hippocampi. In 1735, Duvernoi located the head of the hippocampus in the uncus. The similarity with a ram's horn, cornu ammonis, was noted in 1742 by Wilson. Haller, 1762, saw in the hippocampus an animal's cloven foot and in the uncus he described a large upper and a small lower foot. The smaller foot later became associated with the calcar avis. Lewis also presents a figure showing the similarity between a dissection of both hippocampi of the brain and a statue of a rearing hippocampus. The unci form the two raised feet of the horse.

Of the two names originally suggested by Arantius, Lewis is of the opinion that the Basle anatomic nomenclature has sanctioned the less descriptive but more euphonious one.

GRAY, Chicago.

DYSTHYMIC CHILDREN. SANTE DE SANCTIS, *Encéphale* **18**:1, 88 and 156, 1923.

The rarity of periodic psychopathic states in children is fairly well known. In general, not more than 2 or 3 per cent. of manic-depressive insanity occurs in individuals under 15 years of age and in much less proportion in children under 10. The author gives a full description and discussion of periodic psychopathic conditions in children, and emphasizes not only the importance of a differential diagnosis but also an understanding of the pathogenesis. He inclines to the belief that the pathogenesis lies in a disturbed endocrinosympathetic system. The tendency to a remittent course is dependent on crises in the constitutional rhythm, which occur at times seemingly independently, but also may be influenced by infections and intoxications. The visceral symptoms are not uniformly correlated with the type of emotional deviation. This is because each individual has his own personality, which is dependent not only on the structure of the brain, but on the environmental stimuli which have contributed to the development of the personality.

These dysthymic children comprise an appreciable portion of cases of conduct disorders. The author believes that the strictly medical significance of conduct disorder is often unrecognized. In young suicides, prostitutes and criminals a careful history will often reveal mood deviations suggesting a more or less chronic depression or excitation, as the case may be. These cases are incurable. However, the episodes may be controlled by proper mental hygiene and endocrine therapy is often useful.

De Sanctis' contribution is well worth reading.

HYSLOP, New York.

SYNDROMES OF THE INFUNDIBULAR-HYPOPHYSIAL GROUP IN EPIDEMIC ENCEPHALITIS. J. MOUZON, *Presse méd.* **31**:113 (Feb. 3) 1923.

It is accepted in this paper as sufficiently well established, that the regulation of the metabolism of water and fats and also some regulation of genital function are dependent on cells in the floor of the third ventricle, not on the hypophysis. The appearance of polyuria, obesity, and adiposogenital syndromes can no longer be considered exceptional in epidemic encephalitis, since so many cases with these complications have been reported. The gray matter at the base of the third ventricle has probably been involved in the encephalitic process, though no verification by necropsy is available. The hypophysis may become slightly involved as well; there has been evidence of this in swelling of the cells of the hypophysis found in several necropsies on cases of encephalitis. However, no change in the sella turcica, no hemianopia, optic atrophy, acromegaly nor gigantism, have been demonstrated in the reported cases. That is, the only hypophysial symptoms that have been found have been explainable by a lesion of the floor of the third ventricle alone. Furthermore, the polyuria and the adiposogenital syndrome have not been seen together. The symptoms found have tended to clear up gradually, instead of growing worse as is usual with hypophysial lesions.

HUDDLESON, New York.

LOWER HALF HEADACHE (NEURALGIC) OF NASAL ORIGIN;  
GLOSSODYNIA, OTALGIA, NAUSEA, PARAGEUSIA, VERTIGO,  
SCOTOMA, PHOTOPHOBIA, RHINORRHEA AND ASTHMA AS  
ISOLATED RELATED PHENOMENA. GREENFIELD SLUDER, J. A. M. A.  
79:1898 (Dec. 2) 1922.

In 1908 the author described a "lower half headache" (neuralgic) which seemed to be related to the nasal (sphenopalatine or Meckel's) ganglion. The clinical picture of lower half headache when complete consists of pain about the eye, the upper jaw and the teeth, extending to the zygoma and temple, with earache and pain in the mastoid, emphasized at a point 5 cm. behind it. This point is always tender on pressure, although the pain is often temporarily absent. Thence, it extends to the occiput, neck, shoulder, scapula, arm, forearm, hand and fingers. This is the pain, or neuralgic, syndrome. There may, however, be added to this a sympathetic syndrome of sneezing, rhinorrhea, lacrimation and photophobia.

In these cases, there was almost always a recognizable lesion in the district in which the ganglion lay, and the symptoms could be relieved by cocaineization of the ganglion. Later, he found that typical lower half headache could be present without such a lesion and could not be relieved by such treatment. These were cases of sphenoiditis, hyperplastic or suppurative, and he believed that the headache was produced by irritation or inflammation of the nerves which supply the ganglion. Such lesions, of course, are situated central to the ganglion and naturally are not influenced by its cocaineization. Treatment of the sphenoid is successful for these lesions.

NIXON, San Francisco.

## Society Transactions

### PHILADELPHIA NEUROLOGICAL SOCIETY

*Regular Meeting, May 25, 1923*

C. M. BYRNES, M.D., *President, in the Chair*

#### A CASE OF LESION PROBABLY INVOLVING THE THALAMUS AND SUBTHALAMIC REGION WITH SYMPTOMS OF IRRITATION OF THE SYMPATHETIC NERVOUS SYSTEM. DR. C. S. POTTS.

H. S., watchman, aged 65 years, was admitted to the Philadelphia General Hospital April 12, 1923. With the exception of an attack fifteen years before in which he lost power on the left side and from which he entirely recovered, his previous history is unimportant. For several months previous to his present illness he suffered from headache and became easily fatigued.

April 8, 1923, while dressing, he suddenly became dizzy, things became black before him and he fell.<sup>\*</sup> After being helped to his feet he walked to his bed. Later "the left leg became weak and there was loss of feeling in the left arm and hand." He was never unconscious. When admitted, there was complete motor paralysis of the left side, including the face, with a positive Babinski sign and increase in the tendon reflexes. There was loss of tactile, pain, temperature, pressure, position and vibration senses on this side with left lateral homonymous hemianopia. He perspired freely on this side. The left pupil was dilated and the left palpebral fissure somewhat wider than the right. Cocaine dilated the left pupil but had no influence on the right. Neither pupil reacted to light, nor in accommodation or convergence. These symptoms have persisted with no change except that he occasionally complains of pain on the left side. At times the pupils are more nearly equal and they respond slightly to light. The palpebral fissures are equal in width. The sweating is more profuse at times than at others, but always involves the entire side. The bed clothes are frequently wet from it.

I believe that the lesion is a thrombosis probably involving the posterolateral ganglionic arteries, which are branches of the posterior cerebral artery, and supply the posterior part of the thalamus, the geniculate bodies, corpora quadrigemina, quadrigeminal brachia and cerebral peduncle. They are end arteries, and this accounts for the lack of improvement.

There is considerable uncertainty concerning the relation of the brain to the sympathetic system. Some experimental work, which has been summarized by Spiller in a paper published in the *American Journal of the Medical Sciences* in 1920, points to the existence of a center in the hypothalamus, possibly the hypothalamic nucleus. Such a center could be irritated by a lesion in the probable location in this case.

#### A CASE OF HEMIHYPERTROPHY AND HEMIHYPERTONIA DEVELOPING AFTER TYPHOID FEVER. DR. MARION H. REA.

A colored girl, aged 16, was admitted to the Woman's Hospital, complaining of "weakness and stiffness of her left side." This condition began six years

before, following a severe attack of typhoid fever. She was in bed for three months and when she began to convalesce was unable to walk; the arm and leg were stiff, and speech was also affected. She slowly learned to walk and talk but the left side has since always been weak. The family and personal history, with the exception noted, are of no importance.

General physical examination reveals nothing of note.

*Neurologic Examination.*—The left arm and leg are held in a constrained fashion, the arm straight down against the side and thigh, but in no way suggestive of a hemiplegic arm; the leg is held stiffly, with the foot everted in walking. The outstanding feature is the intermittent hypertonicity of the left arm and leg, left gluteal and left thoracic muscles. There is no true contracture or paralysis, but the recurrent hypertonicity is so great that a simple movement, as putting out the hand, cannot always be performed. She seems to make great effort to comply but cannot; in a few seconds the tonicity diminishes and she does as asked. Excitement increases the tonicity, but as she grows accustomed to the examiner the rigidity practically disappears; at times she can aid in the relaxation; suggestion is also helpful. The second outstanding feature is the hypertrophy of the muscles of the involved side, especially of the deltoid, biceps, forearm, gluteal, thigh, gastrocnemius and thoracic muscles. The last have produced a left dorsal scoliosis. A lumbar kyphosis also occurs during the phase of tonicity. The left foot is wider and shorter than the right; the left hand is smaller and more delicately shaped than the right. On extension there is hyperextension of the fingers and a suggestion of athetoid movements of the fingers. In the face there are irregular convulsive movements slightly suggestive of Sydenham's chorea. Her speech, at times, is difficult to understand; sometimes it is stuttering, sometimes blurred. The reflexes are normal.

I think the hemihypertrophy and hemihypertonia are due to a lesion of the right lenticular nucleus.

#### DISCUSSION

DR. CHARLES K. MILLS: What especially interests me in all these cases is the absolute evidence of the distinct separation of tonectic apparatus from the pyramidal motor system. This tonectic system is, I believe, both cortical and striatal, and has extensions to the subthalamus, thalamus, nucleus ruber, cerebellum, brain stem and spinal cord. Although the lesion is striatal, the symptoms in a certain sense may be cortical. That is, a destructive lesion in the striatum may cause movements which are really aberrant cortical movements.

DR. WILLIAM G. SPILLER: This case is especially interesting in two respects. The symptoms developed after typhoid fever. I have seen a number of cases of hemiplegia which developed during convalescence from typhoid. The condition probably is thrombotic; in one case I found thrombosis in the cerebrum. Dr. Rea's case is one of postapoplectic hemihypertonia of v. Bechterew, and is probably lenticular in origin. The spasm varies from moment to moment, as is characteristic of this condition; it is a mobile spasm.

DR. ALFRED GORDON: Several years ago I observed a man aged 29, who presented, after an apoplectic insult, rigidity and weakness on the left side of the body. The hypertonicity was marked and increased when the patient attempted to perform the least movement; even when he was approached with the purpose of performing passive movements. If I attempted to touch his hand, it would turn and become rigid. I think this is an extrapyramidal involvement.

DR. A. M. ORNSTEEN: I should like to ask Dr. Rea whether the patient had a real hemiplegia during convalescence from the typhoid fever, or just a hemiparesis. Most of these patients have apoplectic manifestations in the form of a hemiplegia, but sometimes the hemiplegia may be so transitory as to escape notice, and the hypertonic manifestations are thought to be the first evidence of involvement of the cerebrum.

A CASE OF ACTINOMYCOSIS OF THE CEREBRUM. DR. ROLAN N. KLEMMER.

A white man, aged 38, a tailor since the age of 8, gave no history of illness until 1914, when a thoracotomy was done on the left side and much "very foul-smelling, thick viscid, greenish pus" was liberated. The pus was sterile on two examinations, but contaminating organisms were found on two other occasions. Three years later a perineal abscess was opened.

He was finally readmitted in November, 1922, for a painful swelling of the right wrist and pain in the lower lumbar region. He was a chronic alcoholic, was poorly nourished and was underweight, presenting the typical appearance of chronic tuberculosis. The spine was fixed from the twelfth dorsal vertebra to the sacrum. Motion in the right wrist became normal in several weeks. Four weeks after admission, a rapidly enlarging painful mass was found in the left hypogastric and lumbar<sup>4</sup> regions, causing flexion of the left thigh. On incision, over a quart of thick, greenish pus with a peculiar odor was evacuated. The cavity was so large that its bottom could not be reached when the entire hand was inserted into the wound. Dr. J. C. Small, of the hospital laboratory, reported a pure strain of actinomyces in the pus, the first indication of the true nature of the disease. Three weeks after operation the discharge had practically ceased, the patient felt better and was able to walk about the ward.

January 21, 1923, after an attack of "cramps" the night before, he showed partial paralysis of the fingers of the right hand with slight impairment of tactile sensation over the right side of his face. During the next three days he had one daily convulsion, lasting from five to twenty minutes, of the jacksonian type, starting in the right hand and extending to the right side of the face, and finally to the right leg. The paralysis meanwhile extended until he had marked weakness of the right side of the face, right arm and right leg. Potassium iodid, 20 grains (1.25 gm.), three times a day, was given because we suspected actinomycotic cerebral metastasis. Four days after the onset, he had occasional twitching movements of the right side of the face and right hand; touch was slightly impaired in the same distribution; there was distinct loss of sense of position in the right hand and right foot, and a positive Babinski sign on the right; but pain sense was normal on both sides. Dr. George Wilson confirmed the diagnosis of cerebral actinomycosis and advised operative exposure of the left motor cortex and parietal lobe. Dr. Laplace believed the infection was progressing too rapidly for operative intervention.

There was slight haziness of the optic disk but no true optic neuritis. Six days after the onset, the patient developed motor aphasia, but was cooperative. The next day he became word deaf; and the following day was comatose. He died nine days after the appearance of the first symptoms indicating cerebral involvement.

The spinal fluid, five days before death, showed increase in globulin and 77 cells per c.mm.; it was clear and not under pressure. The Wassermann test

was negative; the colloidal gold curve was 2223310000. His temperature never rose above 100 F., except for several days during his first week in the hospital and immediately before death, when it reached 101 F.

The necropsy revealed an abscess cavity in the retroperitoneal region of the left side, extending from the twelfth dorsal to the fourth lumbar vertebra. The sides of the vertebrae were exposed, but there was no gross evidence of erosion of the bones themselves. The abscess cavity had been well drained by the operative wound; the walls were collapsed, tough and of a grayish yellow color. Microscopically, they showed old fibrous and new granulation tissue with slight round cell infiltration.

The skull bones, dura and pia mater, with the exception of a very small portion of the latter, were normal. There was distinct bulging with fluctuation of the whole middle portion of the left hemisphere, centering about the middle of the parietal lobe. A slight purulent exudate was found along several of the veins, spreading out slightly over the cortex. Smears and cultures of the sub-arachnoid exudate were negative. Section through the hemispheres disclosed an abscess occupying the left corona radiata and part of the corpus striatum, although not invading the internal capsule. The contents were grayish green, semi-viscid, and had the same peculiar odor as the abdominal abscess, described by Dr. Small as a butyric acid odor. The walls of the abscess were distinct, granular and showed some small, yellowish bodies. Smears revealed innumerable small clumps of the branching filaments of actinomyces—exactly similar to those of the abdominal abscess. Culture on various mediums and animal inoculation were negative. No ray fungi were found in histologic section of any part of the body. Both lungs showed terminal bronchopneumonia and in one section a picture similar to old chronic tuberculosis.

This case, I believe, was primarily a disease of the lungs, of the pleuropulmonary type, resulting in empyema eight years before death. The peritoneal abscess may or may not have been actinomycotic. Finally there was a retroperitoneal abscess and cerebral metastasis.

#### DISCUSSION

DR. GEORGE WILSON: Dr. Klemmer's case was a clear instance of a lesion involving the left motor and parietal lobes. The type of attack was that of *epilepsia partialis continua*. While an operation would probably not have saved his life, it seemed to me that at various times when I examined him the indications were clear and urgent. I should like to ask Dr. Klemmer what has been the result of surgical interference in actinomycosis of the brain.

DR. KLEMMER: I have found only one patient who remained alive for more than three or four days after operation. This case was also one of localized abscess and, as I remember it, the patient lived for a year after operation. There was a recurrence and the patient died several weeks after a second operation.

#### DO FIBRILLARY CONTRACTIONS ALWAYS DENOTE ORGANIC CHANGE IN THE NERVOUS SYSTEM? DR. A. J. OSTHEIMER.

This patient was in the army from May 14, 1918, until January 31, 1919, but his service was entirely in this country. Practically, this period was spent partly in quarters and mostly in hospital, from which he was finally discharged on surgeon's certificate of disability, the diagnosis being a psychosis, manic-depressive in type.

The salient factors in the history are: One brother is deaf and dumb and has been subject to epileptic attacks. The patient had "nervous breakdown" a few years before he was drafted. Examination of the man in August, 1921, showed twitchings of muscles all over his body, in addition to a fine tremor of the hands; there was a good deal of mental retardation and some depression. Psychometric examination in January, 1922, gave the man an intelligence quotient of 64, putting him into the high grade moron class. The Wassermann reaction of the blood has always been negative with various antigens.

Physical examination shows a poorly nourished man, aged 34, with generalized muscular wasting, more marked in the upper extremities. This wasting is symmetrical and there seems to be no real atrophy. It is true that there is a slight difference in size in the greatest circumference of the legs, the right measuring  $12\frac{1}{2}$  and the left  $12\frac{3}{4}$  inches. Three measurements at different levels of the thighs give the following results: right, 14, 16, and 17 inches; left, 14,  $16\frac{1}{2}$ , and  $17\frac{1}{2}$  inches. However, the existence of varicose veins on the left side may account for this difference.

The man's attitude is one of dejection and depression, coupled with an expression of timidity and fear. Fibrillary contractions are prominent and appear occasionally in almost all muscles of the body, except those of the face and neck; they become worse and more frequent as the examination progresses. In addition, there is marked tremor, particularly of the hands and fingers, but also of the thigh muscles and elsewhere. During the examination the man weeps quietly. There is swaying with the eyes closed and slight incoordination of the upper extremities. His gait is slightly spastic, with some dragging of the left toes. There is some rigidity and contraction of the muscles of the upper and lower extremities, particularly marked in the lower extremities; the knee jerks are quick and much exaggerated, while the deep reflexes of the arms are not. There is no inequality of the tendon reflexes. The abdominal and cremasteric reflexes are normal, and while it is somewhat difficult to test the plantar reflex on account of hyperesthesia, it is my opinion that plantar irritation undoubtedly causes flexion. There is a fairly well sustained ankle clonus on the left side with indication of the same on the right, and at times patellar clonus can be elicited on either side. The pupils are middle wide, equal, regular in outline and react promptly to light and in accommodation. There are practically no disturbances of sensation or motor power, except as noted. There is no astereognosis nor disturbance of the sense of position. The hands are cold and clammy. Electrical reactions of the muscles were found to be quite normal on April 28, 1923, and there is at present no disturbance of idiomuscular contraction. Mental examination elicits nothing more than a condition of depression, with general mental retardation and dulness, which may very well be indicative of his mental defect.

The case is puzzling, because, notwithstanding the fact that there are marked and definite signs such as are usually attributed to organic nervous disease, it would still seem that, in the absence of a Babinski reflex, it might be possible to explain all signs on a purely functional basis. The facts that the electrical reactions are normal, and that the fibrillary twitchings have existed for over two years without real progressive atrophy seem to rule out all forms of progressive muscular atrophy of spinal origin. From the standpoint of the underlying abiotrophy, and from the easily discernible large psychoneurotic factor, I am inclined to believe that the entire condition is functional.

French writers have described a fibrillary chorea, a condition characterized by fibrillary contractions first appearing in the muscles of the calves and of the posterior portion of the thigh, extending to the trunk muscles and even to the muscles of the arms and shoulders, but never to the muscles of the face and neck. These contractions do not involve the entire muscle, but only individual fiber bundles, and result only in slight elevations of the skin. This so-called fibrillary chorea of Morvan is not considered a serious disease, although it may continue over a long period of time with frequent fluctuations.

In this connection, I would call attention to the fact that Dejerine believes that the tendon reflexes may be much exaggerated and that an ankle clonus can exist in hysteria. One may conclude, therefore, that patellar clonus can be due to the same underlying condition. Even the slight indication of spastic gait in this case I believe might be consequent on this same functional factor.

#### DISCUSSION

DR. WILLIAM G. SPILLER: I do not believe fibrillary tremor is necessarily indicative of organic disease; while of great value in muscular atrophy, I am convinced it may occur as a sign of functional disorder.

DR. CHARLES S. POTTS: If the fibrillary tremors in this patient are due to organic disease, there should be more atrophy. His muscles seem fairly well developed. I do not think a man can simulate fibrillary tremors, even with compensation as an object. In tuberculosis and other conditions that cause emaciation one may see fibrillary tremors. It seems to me more like a functional than an organic case.

About ten years ago I presented before this society, a normal man who had persistent ankle clonus. He was a medical student, is still normal and still has ankle clonus. A number of years ago I reported a case of exophthalmic goiter in which ankle clonus was present, which disappeared as the exophthalmic goiter improved. A paper has been published by Tileston in which he mentions a number of causes, not organic diseases of the nervous system, for ankle clonus; many were toxic disturbances.

DR. GEORGE WILSON: I have examined this man and believe that he has a psychoneurosis, and that the fibrillary contractures are of functional origin.

DR. ALFRED GORDON: I believe that the man has some atrophy. When he performs movements with his arms, atrophy is evident in individual muscles. It is true that he has a flexor plantar reflex, yet the case suggests organic disease, although the tremor may be functional.

DR. J. HENDRIE LLOYD: This man was in the army from May, 1918, until January, 1919. He spent most of that time in the hospital and was never exposed to danger. I do not know to what his fibrillation is due, but as he has no atrophy, it cannot be due to anterior poliomyelitis. I think his tremor is characteristically hysterical. It has the very fine rhythm that is seen in hysterical tremor. His general attitude is that of a moron, as Dr. Ostheimer has pointed out, and he is on the list of disabled veterans. He will probably never get well as long as he is paid for remaining as he is.

DR. CHARLES K. MILLS: I am rather inclined to disagree with the opinions expressed to the effect that this is a "pension tremor." This man's syndrome has existed for two years. I do not believe there was ever a case of true fascicular tremor that was purely functional. I have seen cases much like this, quite independently of any hope of a pension. Years ago, I reported a case,

undoubtedly organic, under the name of generalized undulatory tremor. How this man can in any way produce a tremor of this kind is impossible of comprehension to me. Of course men have shivers in coming out of battle or in any desperate situation, as mentioned by Dr. Wilson, but this is different and does not persist for two years.

I recognize no case as hysterical in which the possible intervention of the controlling cerebrum is not present. Dr. Spiller referred, in connection with this case, to the question of the ankle clonus. There is no such thing as persistent ankle clonus with a Babinski reflex in pure hysteria, although a pseudo or abortive clonus may be present in spastic hysteria. As Dr. Potts has said, no one, even for an increase in his compensation, can simulate a tremor of this kind. When we speak of an organic condition, we do not necessarily believe in an isolated focal lesion of some sort. Many of the cases that are classed as functional are toxic and I regard a true toxic condition as organic, although it may be only temporarily so.

#### SPECIMENS OF BRAIN TUMORS REMOVED AT OPERATION. DR. OGLESBY.

These three frontal lobe tumors were removed at operation by Dr. Charles H. Frazier in the course of nine days. They are of interest because of the variation in the duration of symptoms: two years and eight months, nine months, and nine weeks, respectively. Convulsions occurred in two cases, and in two the primary symptoms were disturbances of vision. Unilateral exophthalmos was noted in two cases. It subsided after operation. In one case the intraspinal pressure was not increased. One patient developed a psychosis—was delirious, noisy, and required constant restraint before her first stage operation.

CASE 1.—A man, aged 25, was admitted to the hospital Oct. 10, 1922, complaining chiefly of blindness. In 1919 he first experienced a tired feeling when he read. In September, 1920, he was admitted to the ophthalmologic service of the hospital and was found to have a positive blood Wassermann reaction and choked disks of 5 diopters. He received antisyphilitic treatment. In May, 1921, his vision failed suddenly; he had had severe headaches for a few months previously. Subtemporal decompression was done and relieved the headaches, but vision was soon completely gone. He had attacks of paresthesia on the right side of the body and face, twitching of the right eyelid, and would lose his balance.

The results of examination were negative except for diminished patella reflexes and choked disks of 5 diopters. A roentgenogram showed some enlargement of the sella—the basal metabolic rate was plus 6. Nov. 6, 1922, a left parietal flap was laid back, but nothing was found. Nov. 11, 1922, a ventriculogram showed a frontal lobe tumor, but further operation was deferred because of wound infection. Feb. 5, 1923, anosmia and impairment of taste were present; the knee jerks were exaggerated and he was apathetic. March 23, 1923, a convulsion occurred, general at first, but, later, only on the right side. An operation was performed April 2, 1923, and a large subcortical tumor was removed from the right frontal lobe; it extended to the base, but its origin was undetermined; its weight was 210 gm.; histologically, it was a meningeal endothelioma.

CASE 2.—A white woman, aged 31, complaining of vomiting, headache and failing vision, gave a history of a sudden onset four weeks before. Within a

few days the vision of the right eye began to fail, and this progressed rapidly until she soon became practically blind in that eye. Vision of the left eye also failed gradually. She had no convulsions, and no motor or sensory disturbances were present.

She had a left exophthalmos, and choked disks of 4.5 diopters on the right and 2.5 diopters on the left. The visual fields showed left homonymous hemianopia with constriction of the upper part of the fields. Complete anosmia was present and distinct weakness of the right side of the face. The tendon reflexes on the left were increased but a Babinski sign was not obtained. No muscular weakness was evident. The roentgenogram was negative. The intraspinal pressure was 40 mm. Ventricular puncture was done, and 15 c.c. of fluid was removed, but no air was injected. The patient later developed a psychosis.

April 6, 1923, a first stage operation was done—the dura was under tension. A right frontal flap revealed a tumor which was not removed. The psychosis became more marked and the general condition of the patient grew progressively worse. April 27, 1923, the second stage of the operation was performed and a large tumor occupying the greater part of the right frontal lobe and extending across to the left side was removed. The patient succumbed.

CASE 3.—A white woman, aged 55, was admitted April 27, 1923, in a condition of stupor. In July, 1922, she had made extraordinary mistakes while playing cards and had talked irrelevantly. About noon she fell in a series of convulsions that involved all four extremities, and were followed by vomiting. She remained in bed for ten days at this time.

The results of examination were negative except for increased reflexes. Feb. 3, 1923, she had another general convulsion with incontinence of urine and biting of the tongue, followed by somnolence for two days and transient mental confusion. April 3, 1923, she was taken acutely ill with headache; she was irrational and failed to use the proper words. She tottered when she walked and had tremors of the right hand. April 17, 1923, she gave meaningless monosyllabic answers to questions. The tremor and weakness of the right hand with increased reflexes on the right side persisted.

She entered the Howard Hospital Feb. 19, 1923. Moderate choked disks were discovered April 23, 1923. She yawned a great deal, was unable to walk, had impaired sense of smell on the left, was semistuporous and apathetic but appeared to understand questions. The left pupil was the larger and reacted sluggishly. Ptosis of the left lid with left exophthalmos was evident. Increased reflexes on the right with convulsive movements of the right arm and tremor of the hand were noted with a Babinski sign on the right.

April 27, 1923, the white blood cells numbered 21,000; the spinal fluid and blood were negative. The eye grounds showed choked disks of from 2 to 3 diopters with hemorrhages on the left side.

May 1, 1923, a left frontal flap operation was performed and an encapsulated tumor was easily removed from the surface of the frontal lobe.

#### DISCUSSION

DR. J. W. McCONNELL: It was especially noteworthy that the active symptoms of neoplasm in Case 3 progressed so quickly. In the last several years I have had under my care five cases of frontal tumors, and in only one were the symptoms of a duration sufficiently long to permit a definite localization. One man with a tumor practically filling the right frontal lobe had been working as a railroad conductor up to the night he was taken sick. His first symptoms

were nausea and vomiting supposed to be due to acute indigestion. He died suddenly within ten days after admission to the University Hospital. In these cases the antemortem diagnosis was not made sufficiently early to be of any value, as they showed no localizing symptoms whatsoever. I saw Dr. Oglesby's third patient recently and she now can play bridge-whist, walk and talk; and from every standpoint is practically normal.

DR. WILLIAM G. SPILLER: When a frontal lesion develops slowly it may not produce the same symptoms as when it develops rapidly. In Dr. McConnell's patient the stupor before the operation was profound, and it was almost impossible to get the woman to obey the simplest command. Within a week or so following the operation her mentality was so good that her husband could recognize no mental failure. The tumor was an endothelioma and caused atrophy of the frontal lobe sufficient for its accommodation, and although the tumor was large the function of the frontal lobe was not disturbed when the pressure from the tumor was removed.

Another interesting feature of the case was intention tremor only in the right upper limb.

DR. CHARLES K. MILLS: The presence of few symptoms until late in the disease is not uncommon in slowly growing endotheliomas and is not without explanation. The growing neoplasm very slowly compresses, but does not destroy the centers or the tracts beneath it, and these continue to function. When the endothelioma is removed, the brain slowly recedes to its original position and largely regains its original functions. Something more or less similar occurs in hydrocephalus in which compression of the brain occurs from within outwards.

#### A CASE SHOWING EVIDENCE OF MUSCULAR DYSTROPHY AND PITUITARY DISTURBANCE. DR. C. A. PATTEN.

This patient, a boy, aged 12, is presented because of the rare coincidence of a typical Fröhlich syndrome and pseudohypertrophic muscular dystrophy. He was a full term child with normal birth and had no illness until he was 4 months of age. At that time he had pertussis followed by double pneumonia and was gravely ill. After that he was weak and did not develop well for several years. At the age of 2 years he began walking, but with difficulty. He fell frequently, usually forward, occasionally backward. Until the age of 6 years, he was unable to ascend stairs alone except by grasping the rail and pulling himself up hand over hand. He has never been able to run and play like other boys, because of definite muscular weakness. If he fell to the floor he always had difficulty in rising, usually pulling himself up by some nearby object, or else climbing up himself. The hands also were weak. At the age of 10 he weighed 40 pounds and was very thin. At this time his weight increased rather suddenly and in the past two years he has gained 35 pounds. With the gain in weight there has been increased difficulty in the use of his muscles. He has always been a quiet, well behaved boy, and has reached the fifth grade in school. This school record would indicate, perhaps, some mental retardation. The family history is negative throughout.

Examination shows a short boy with considerable excess of fat, segmentally distributed. The hair on the head is abundant and rather coarse. The genitalia are infantile and are buried in pads of fat. The pulse ranges from 82 to 112. Basal metabolism is minus 13 per cent. All serology is negative.

Blood chemistry examination revealed: non protein nitrogen 34 mg.; urea nitrogen 13.4 mg. per 100 c.c. of whole blood. Urinalysis was negative. A sugar tolerance test gave the following figures: 97.1, 111, 102.7 and 100 mg. per 100 c.c. of whole blood. This curve, which is not indicative of pituitary deficiency, is probably explained by the fact that before coming under observation he had been given pituitary extract for a period of some months. A roentgenogram of the sella showed no abnormalities, the anteroposterior diameter being 5 mm. and the depth 7 mm. A roentgenogram of the chest showed no thymus shadow.

There seems to be a large possibility of endocrine origin for the metabolic disturbance in the muscular dystrophies. In this case there is a history of muscular difficulty from infancy and as the boy neared the age of puberty, the time when the endocrine system together with all other tissues and organs of the body undergo great changes, there has developed an excessive deposit of fat, and a failure of genital development, which places the individual in the hypopituitary class. In addition one should always consider the possibility of thymic subinvolution in these cases.

#### DISCUSSION

DR. ALFRED J. OSTHEIMER: When I visited Babinski in 1919, a case of this kind was examined, and Babinski said that he had described and published his paper on this condition a few months before Fröhlich's article appeared.

#### CLINICO-PATHOLOGIC REPORT OF AN UNUSUAL CASE OF ENCEPHALITIS. DR. N. W. WINKELMAN.

The patient B. C., a white woman, aged 54, was brought to the hospital May 4, 1923, by the police who found her wandering on the streets. She was much confused and was unable to give a coherent account of herself. The history obtained from her brother stated that she was working as a shirt-maker up to the day she was taken to the hospital, but had not been well for about three weeks. During this time she had complained of double vision; her speech was thick and she had weakness of one side of the face. She had had both breasts removed; the first in February, 1918. Microscopic examination showed it to be a "scirrhous carcinoma simplex of the breast." The other breast was removed a few years later. Examination revealed an emaciated, disoriented, and confused woman. The operation scars were completely healed and there were no local recurrences, though enlarged glands were felt along the carotid vessels and in the supraclavicular spaces. The pupils reacted slowly. In walking she always staggered to the left; there was a Babinski sign on the left. While in the hospital she had a series of generalized convulsions; there were twitchings of the right side of the face and a peripheral left facial paralysis. Spinal puncture showed no increase of pressure; the fluid contained blood. The patient gradually became more stuporous and died eight days after admission, the twitchings of the right side of the face continuing to the end.

Necropsy revealed gross petechial hemorrhages, limited to the left side, in the face and arm center of the brain extending backward into the parietal lobe, with another similar area in the right frontal pole. Microscopically, the petechial hemorrhages were evident not only in these areas, but also to a much less degree, in the caudate nucleus and in the cerebellum. There was mild yet distinct perivascular infiltration of round cells in the basal ganglions, midbrain, and pons and, to a much less degree, in the medulla. The substantia nigra was not involved. The smaller vessels were packed with and some

occluded by cancer cells. This was especially evident in the neighborhood of the hemorrhage but was also present to a less degree throughout the nervous system.

There were metastatic nodules of considerable size in the lungs and one rib was also involved.

This case was one of generalized carcinomatosis together with an infection. In one place only was it found that the cancer cells had broken through the blood vessel walls and were invading the nervous tissue. The reaction around the vessels in the basal parts of the brain was probably part of an encephalitic process, not necessarily of the epidemic type. The petechial hemorrhages were in all probability due to blocking of the vessels by plugs of cancer cells, together with local changes in the vessel walls. The *epilepsia partialis continua* was attributed to the localized petechial hemorrhages in the left facial center.

## Book Reviews

### THE FORM AND FUNCTIONS OF THE CENTRAL NERVOUS SYSTEM. AN INTRODUCTION TO THE STUDY OF NERVOUS DISEASES.

By FREDERICK TILNEY, M.D., Ph.D., Professor of Neurology, Columbia University; Attending Neurologist, the Presbyterian Hospital and the New York Neurological Institute; Consulting Neurologist, Roosevelt Hospital, New York, and HENRY ALSOP RILEY, A.M., M.D., Associate in Neurology, Columbia University; Associate Attending Neurologist, New York Neurological Institute; Attending Physician, Neurological Department, Vanderbilt Clinic, New York, with a Foreword by GEORGE S. HUNTINGTON, Sc.D., M.D., Professor of Anatomy, Columbia University. Cloth. Second Edition. Pp. 1,019 with 763 illustrations, of which 56 are colored. Price \$12. New York, Paul B. Hoeber, 1923.

The advent of this book marks a distinct epoch not only in the teaching of nervous anatomy and physiology but also in the evolution of medical training in general. It presents in an eminently successful manner the answer to the constantly growing demand for a real coordination between preclinical and clinical branches. To the average medical student, the study of neurology has always appeared as a dry and uninteresting task requiring a supreme effort of almost pure memory. The signs and symptoms have appeared arbitrary and confusing, the different diseases seeming to be compounded without plan from various combinations and permutations of a number of formal and unrelated factors. His recollection of nervous anatomy has usually been a more or less confused jumble of lobes, fissures, nuclei, ganglions, ventricles and tracts. His memories of physiology have been largely of isolated "preparations," electrical changes, stimuli and inhibitions which have seemed to be without guiding purpose or plan.

The authors of this book have undertaken the task of bringing purposeful order out of the seeming chaos and of presenting the nervous system as a living and functioning unit in such manner that "the anatomy and physiology of the central nervous system are no longer permitted to remain as independent branches of medical science, but are here incorporated as essential parts of the practical knowledge necessary to the proper diagnosis and treatment of disease." The complexities of structure are elucidated from embryology and comparative anatomy, the practical bearing of the various nervous connections being illustrated by clinical histories and pathologic reports of cases in which particular paths have been damaged by disease or injury. The inclusion of clinical syndromes serves not only to bring about a clearer understanding of the anatomy and physiology by the use of concrete illustrations but also to establish the importance of neurology to the practicing physician.

After a clear, concise and well illustrated review of the general embryology and a description of the neurons and their integration into physiologic mechanisms, the component parts of the nervous system are studied in detail. The subdivision is made into spinal cord, medulla, pons, cerebellum, midbrain, interbrain and endbrain. Each is described as it exists in situ with its relations to other structures, its blood supply, its histologic characters, the functions of its neuron systems and the clinical syndromes that result from damage in various localities. These last are illustrated profusely with reports of actual cases.

The illustrations are numerous, many of them from original work by the authors and their co-workers, and are beautifully executed, some of them in colors. The diagrams are admirably clear. The statements concerning function are conservative and adhere strictly to demonstrated facts; when the facts are still in doubt, helpful suggestions have been offered as to the most probable interpretations. The value of the book is greatly enhanced by a glossary of terms, copious references for supplementary reading and an excellent index.

The book is the culmination of extensive research in embryology and comparative anatomy, much practice in teaching and a rich clinical experience, combined with a happy facility for clear expression. It will probably be objected that the book is too large for the general student, but it is difficult to see how it could be abbreviated without sacrificing the principal object with which it was prepared. The book will be in use throughout the whole period of the medical course, and the importance of a fundamental knowledge of the nervous system for medical practice cannot be overestimated since it serves as the coordinating and controlling agency for all activities of the body. The student who proposes to specialize in neurology will find here a solid foundation on which to build, and teachers, both clinical and preclinical, will obtain both inspiration and instruction from its perusal.

H. OPPENHEIM'S *LEHRBUCH DER NERVENKRANKHEITEN FÜR AERZTE UND STUDIERENDE*, By R. CASSIRER of Berlin, K. GOLDSTEIN of Frankfurt-am-Main, M. NONNE of Hamburg and B. PFEIFER of Halle-Nietleben. Volume I, Seventh Enlarged and Revised Edition. Cloth. Price, 27 marks. Pp. 923 with 323 illustrations and 4 tables. Berlin, S. Karger, 1923.

Oppenheim's "Lehrbuch" has so long been accepted as a standard authority that it needs no introduction to neurologists in any country. During the ten years that have elapsed since the sixth edition of this, the first, volume was published, neurology has made such notable advances, many of them the outcome of experience arising in the war, that works of reference that appeared before that time have become largely obsolete. Unfortunately, Hermann Oppenheim, who died May 23, 1919, was not spared to evaluate and incorporate this newly acquired knowledge with that of the past. Faced with the alternative of allowing this magistral work of the genius of Oppenheim to pass into history, the heavy and responsible task of revision was undertaken by his pupil and colleague Cassirer, who was fortunate in enlisting the assistance of Goldstein, Nonne and Pfeifer. These names, in themselves, are sufficient guarantee of a high standard of accomplishment.

The revision has been carried out with the express purpose of making as few alterations in the original text as was consistent with bringing the work up to date; many of the changes consist in the addition of references to literature of the last decade with such modifications of the views expressed as were rendered necessary by the researches reported. These references have been selected with the same broad, cosmopolitan outlook that was so characteristic of the earlier editions. The illustrations have been enriched by a number of excellent plates, some additional and some replacements, which are mainly roentgenograms or illustrations of pathologic material.

The largest additions to the text deal with the results of injuries to the spinal cord and peripheral nerves which occurred in such numbers during the war. Under the heading of anatomy and physiology there have been incorporated paragraphs dealing with recent developments in our knowledge of the

extrapyramidal motor mechanisms, knowledge that has largely accrued from the observation of epidemic encephalitis and its sequelae and the extensive researches on decerebrate rigidity. Considerable space has been devoted also to the trophic and vasomotor functions of the nervous system, the pilomotor reflex work of Thomas and the vegetative functions of the body. In the section on special diseases, reference is made to the investigations of Richter on tabes, and of Flexner and Noguchi on poliomyelitis, to the pathology of myelitis, multiple sclerosis and psuedosclerosis, and to the surgery of spinal cord tumors, and an excellent review is given of the effects of and differences between conus and cauda equina lesions.

In spite of the rigid retention of the original wording with the substitution of the name of Oppenheim for the personal pronoun I, when reference is made to his work and observations, the text reads smoothly and does not offend, as might be feared, with an appearance of patchwork. Cassirer and his co-workers are to be congratulated on the manner in which the revision has been carried out, which admirably maintains the high standards set by Oppenheimer. The appearance of the second volume, in which even larger modifications are to be anticipated, will be awaited with great interest.